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PREFACE

It is the duty of an editor of a work such as the present to accept the responsibility for disappointments and delays and to hold his tongue. As, however, in the case of the second volume, which was sadly delayed by the tardiness of the Vaccination Commission, so perhaps the delay of the present volume may be attributed to causes beyond the control of editor or contributor. An article by Professor Welch on "Thrombosis and Embolism" was to have been in my hands in ample time for the publication of this volume last Christmas. With this exception the volume was ready for press some weeks before that season, and an explanation is due not only to our readers, but also to the several authors whose work was thus long before its appearance put beyond the possibility of revision. Unhappily, before the completion of his task, Professor Welch found himself committed to a desperate fight for the freedom of physiological research in the United States. As President of the Congress of American Physicians and Surgeons, the duty fell upon him of organising the profession in opposition to a bill, now before Congress, charged with restrictions so vexatious that all enlightened medical and scientific men in America consider that its enactment would be nothing less than a calamity. In addition to his laboratory and lecturing work Dr. Welch had to write innumerable letters, prepare papers and documents, organise committees, visit Washington repeatedly, and have interviews with senators. He is unkind enough to add that "all this trouble comes from England, where, if your scientific men had made any sort of courageous stand twenty years ago I should not have been sacrificing my time and energies in this way. I am disposed to think that the trouble I have caused you is a judgment for the delinquencies of your countrymen in this matter." Such are the uncertainties in the course of political business, that although Professor Welch expected every week that the matter would come to the vote, and thus liberate him to fulfil his obligations to me, yet week after week he has been disappointed, and compelled to keep up agitation and organisation. Even yet the fate of the bill is not sealed. Had Professor Welch foreseen this grievous delay he would have withdrawn from his engagement in time for me to place the article in the hands of another contributor. This story I have ventured to tell at some length because it contains an interest and perhaps a warning for us all.

I have now altered the order of the articles, and postponed Diseases of the Arteries to the following volume, at some sacrifice of the natural order of the subjects. Professor Welch's article is one the readers of the *System* would not willingly forgo; I trust, therefore, it will soon come into my hands, and enable me to produce the sixth volume, in other parts ready for press, before midsummer. Dr. Rolleston has been good enough to give me the advantage of his assistance and advice as heretofore, an advantage which, I trust, he will continue to me till this work is finished.

Dr. Mackintosh of Clapham has sent to me the list of errata in the third volume which is printed upon another page of this one; for this kindness I trust that in a sincere and chastened spirit I am duly thankful to him.

THE EDITOR.

CAMBRIDGE, *March* 1898.

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In order to avoid frequent interruption of the text, the Editor has only inserted the numbers indicative of items in the lists of "References" in cases of emphasis, where two or more references to one author are in the list, where an author is quoted from a work published under another name, or where an authoritative statement is made without mention of the author's name. In ordinary cases an author's name is a sufficient indication of the corresponding item in the list.

DISEASES OF THE RESPIRATORY ORGANS

(CONTINUED)

BRONCHITIS
BRONCHIECTASIS
PNEUMONIA
PHTHISIS PULMONALIS
PNEUMOCONIOSIS
PULMONARY ASPERGILLOSIS
EMPHYSEMA OF THE LUNGS
ASTHMA AND HAY FEVER
SYPHILITIC DISEASE OF THE LUNGS

BRONCHITIS

We owe to Badham the introduction of the name bronchitis. Before him and Laennec the disease was confounded with the catarrhs, and more or less with phthisis and pneumonia; from the latter, however, in its broncho-pneumonic form it was distinguished by the name "peripneumonia notha." The name "peribronchitis" is reserved for an affection which chiefly implicates the outer coat; but the distinction is rather one of degree than of kind, the three coats being more or less involved in all cases. The clinical type of the disease is apt to vary with its distribution in the chest, with the degree of its severity, with its course and duration, and with its kind; and additional sub-varieties arise from its manifold associations with other diseases and from the multiplicity of its causes. The size and calibre of the bronchi concerned are also important factors. The patency of the smaller, and especially of the non-cartilaginous tubes, largely depends on a free transmission of the mechanical forces of respiration; that is, on the even and symmetrical play of the surrounding pulmonary tissue. Tubes of minute diameter, whilst easily blocked by tenacious secretion, have little expulsive force for its removal; their inflammation is thus fraught with special consequences. The pathological results of bronchitis are not, however, limited to an interference with the air-conducting function, nor to changes in the mucous membrane; collateral changes may be set up. Bronchitis and bronchiectasis cannot, therefore, be satisfactorily studied in their various aspects without a brief preliminary reference to the anatomy and relations of the bronchial system.

The normal structure and relations of the bronchial tubes. — The distribution of the air-tubes in relation to the pulmonary substance is such that the lobules, which may be regarded as the pulmonary periphery, occupy not only the surface but also the centre of the organ. The perfect and even respiratory movements of the lung, associated with a minimum of pleural friction, are essentially dependent upon a uniform patency of the air-tubes. If the central lobules should fail to expand, compensating stress will fall upon the outer periphery — a result clearly seen in emphysema. The bronchi distributed to the more central parts of the lung being shorter and narrower than those proceeding to the surface may perhaps be more easily obstructed; and in any portion of the lung structural conditions may place some of the tubes at a relative disadvantage. The part which these easily obstructed bronchioles may play in the genesis of bronchiectasis will be explained under that heading. Their temporary obstruction in bronchitis would tend to increase any pre-existing hyperinflation of collateral lobules.

The relation of the bronchi to the pulmonary parenchyma is not merely one of direct continuity; close vascular connections establish a functional relationship between the respiratory surface of the air-cells and that of the intralobular bronchioles. With the pulmonary stroma the connection is also intimate. In each lobule the peribronchial tissue (as well as the periarterial) is continuous with the perilobular tissue, and therefore also with the interlobular connective tissue which binds together all the lobules. Lastly, with the visceral pleura the bronchi present a definite, though more distant, relation. The deep layer of the visceral pleura is nothing more than the perilobular investment of the superficial lobules; and the interlobular septa throughout the lung may be regarded as a continuous prolongation of this subpleural layer. The structure of the bronchi is as follows:—The epithelial lining, consisting of three layers of cells, (*a*) columnar ciliated, (*b*) pyriform, and (*c*) flattened (Debove's membrane), rests, according to Professor Hamilton, on a tough, homogeneous, elastic membrane, the basement membrane, which is pierced only by the wide orifices of the mucous glands. An inner fibrous coat underlies this membrane, and is separated by the muscular coat from the outer fibrous coat in which are embedded the cartilages and the mucous glands.

The adventitia or outer fibrous coat is in intimate relation with the perilobular, and therefore with the intralobular tissue of each lobule. In the case of the larger bronchi the connection is also direct with the interlobular stroma. The adventitia is thus the medium of extensive communications, chiefly lymphatic, between the air-tubes and the rest of the lung; and in disease it shares in all those processes to which the term "interstitial" is applied.

The muscular coat, in addition to those functions which are obvious, may also discharge other physiological duties, a knowledge of which might throw light on pulmonary pathology. Hitherto we have heard more of the perversion of the function of the bronchial muscles than of their natural uses. It is generally admitted that they are liable to tonic spasm, and that this spasm and the resulting partial closure of the smaller air-tubes enter largely into the causation of asthma, and in varying degrees complicate the respiratory difficulties special to bronchitis.

The vascular system of the bronchi consists of the posterior or main bronchial arteries originating from the descending aorta, the anterior bronchial arteries supplied by the internal mammaries, and the small branches contributed by the œsophageal, mediastinal, and pericardial arteries; these vessels accompany the bronchi, supplying not them alone, but the entire pulmonary stroma with nutrient blood, the pulmonary artery being exclusively subservient to respiration. The capillaries of both sets of arteries anastomose freely in the alveolar district, and probably also in the mucous membrane of the air-tubes. According to Zuckerkandl, " only the larger bronchi are irrigated by the bronchial arteries, the terminal tubes being vascularised by the pulmonary artery, and the intermediate bronchi by both."

A similar intercommunication exists between the bronchial veins of the smaller air-tubes (and even, according to Zuckerkandl, of the larger ones) and the pulmonary veins. The bronchial veins also anastomose in the posterior mediastinum with the venous plexus formed by branches from the œsophageal and from the diaphragmatic veins (Hamilton).

The bronchial lymphatics take their origin in the inner fibrous layer, which is in lymphatic communication with the tunica muscularis and, through the thickness of the latter, with the abundant plexus of the outer fibrous layer, where probably they are chiefly discharged into the periarterial channels. Both fibrous layers contain lymphatics in abundance; but since, according to Hamilton, these do not traverse the basement membrane, no absorption would take place from the epithelial lining, and the emunction of the latter would be effected directly into the bronchial lumen.

Before and at its entrance into the lobule the lobular bronchiole is in lymphatic connection with the perilobular and with the interlobular network.

Within the lobule the lymphoid tissue described by Arnold (which also occurs under the pleura) is distributed around the alveolar passage and in the bronchial wall, as well as along the blood-vessels. The peribronchial masses are said to occur on the side of the bronchus opposite to that occupied by the accompanying pulmonary artery.

The activity of the *intra-alveolar lymphatics* is shown by the rapid absorption of the products of pneumonia. The interepithelial spaces and their connective tissue corpuscles communicate with interalveolar plasmatic spaces or lymph capillaries, which converge either into the superficial or into the deep lymphatic network of the lobule. The larger vessels which arise from both these networks accompany the pulmonary arteries and veins to the hilus; whilst another set reaches the latter from the superficial subpleural lymphatic network. According to Hamilton, the subpleural lymphatics have but little intercommunication with the lobular.

In the carbon-injected miner's lung (which usually is not fibrosed) the entire lymphatic scheme is displayed; and this may be studied in Hamilton's beautiful illustrations. According to Hamilton, the soot particles lie in the perilobular and interlobular tissue, around the pulmonary artery and bronchi, in the lymphadenoid bodies of the lung and of the bronchial glands, in the alveolar walls (sparsely), in their epithelial interspaces, and in their desquamated epithelial cells.

The absence of pigment from the visceral pleura might have been expected; its absence from the bronchial mucous membrane (which retains in the miner's lung the pink hue of bronchitis) is explained by Hamilton and others in connection with the impermeability of its epithelium and basement membrane, the injection of the lymphatics taking place through the alveoli only, which but few of the inhaled particles would reach. The isolating property of the basement membrane thus demonstrated has much significance from a pathological as well as from a physiological standpoint.

Classifications of bronchitis.—*The anatomical nomenclature.*—Although a separation of the air-tubes, according to their size, into (*a*) the large bronchi, (*b*) the middle-sized bronchi, (*c*) the small bronchi and sublobular bronchioles, and (*d*) the intralobular or capillary bronchioles, is in great part conventional, still this supplies a convenient anatomical classification for the varieties of simple bronchitis, among which we may describe the following:—

i. Trachea-bronchitis, or bronchitis of the large tubes. = An inflammation of the trachea and larger cartilaginous tubes.

ii. Simple or mild acute bronchitis, or bronchitis of the middle-sized tubes. = An inflammation of the tubes of medium size, and of the smaller cartilaginous tubes.

iii. Severe acute bronchitis of the adult, or acute suffocative bronchitis, or bronchitis of the smaller tubes. = An inflammation of the smallest cartilaginous, and of the non-cartilaginous tubes down to the lobular bronchioles.

iv. Capillary bronchitis of infancy and of old age, or "peripneumonia notha." = An inflammation of the sublobular and lobular bronchioles, extending into the intralobular bronchioles and air-passages.

These groups are not rigidly isolated but frequently combine. Whilst trachea-bronchitis and bronchitis of the middle-sized tubes most often occur independently of the later members of the series, and often indeed independently of each other, the most important of the mixed forms are those involving some of the small tubes in addition to the larger ones.

There is also a *clinical nomenclature* based upon the severity and duration of the attack. Although usually acute in their onset, trachea-bronchitis and bronchitis of the middle-sized tubes are, in themselves, seldom severe or dangerous. Bronchitis of the fine tubes is always a severe as well as an acute affection, and its termination in death or in recovery is usually not long delayed. This special feature of acuteness and of gravity explains, and to a certain extent justifies, the use of the term "acute bronchitis," which has been applied to it by rule of custom, but which is neither exclusive nor explicit. "Chronic bronchitis" is also a general name which has become specialised, without, however, involving much ambiguity; for, neither capillary bronchitis nor bronchitis of the small tubes being susceptible of a chronic development, this name can only apply to group ii., which is exceedingly prone to chronicity, in contrast with trachea-bronchitis which is apt to be recurrent rather than lasting.

The occasional severity of the acute stage of a primary bronchitis of the middle-sized tubes shows the disadvantage of monopolising the name "Acute Bronchitis" as a name for the affection of the smaller tubes; and secondary varieties will be described, which may also run through acute and subacute stages.

Lastly, a *pathological nomenclature* recognises a bronchitis pure and simple—not hitherto traceable, as in the diphtheritic, the tuberculous, and some other forms, to parasitic influences—which presents the following well-marked varieties:—

i. Catarrhal bronchitis: (*a*) simple mucous catarrh; (*b*) chronic or muco-purulent catarrh.

ii. Plastic bronchitis.

iii. Putrid bronchitis.

The immense majority of cases belong to the catarrhal group. The putrid purulent form is seldom met with. The plastic variety is so rare as to be little more than a clinical curiosity.

I. SIMPLE BRONCHITIS

Causation. — A. *Remote causes.*—(i.) No age is exempt; but during early adult life the disease is much less prevalent, in spite of greater exposure. Infants and the aged are particularly liable to it, and the periods of dentition favour its onset. (ii.) Except during the working periods of life, when men are more exposed, sex makes little difference. (iii.) Many occupations involve direct exposure to the extremes of temperature; others are indirect causes, through relaxing influences or confined air. Some trades lead to inhalation of fumes or particles which mechanically set up bronchitis, such as particles of steel, granite, chalk, charcoal, or cotton. (iv.) Luxurious habits both in diet and in clothing, and the overheating of rooms, induce a liability which is especially regrettable in childhood, when the individual tendencies are capable of some measure of control. (v.) Heredity and temperament constitute distinct factors; a delicate bronchial membrane may be inherited as a delicate skin or any other outward peculiarity may be. Again, acquired constitutional weakness from any cause (poverty, overwork, prolonged illness, or intemperance) has an unfavourable effect. (vi.) Certain blood diseases favour the production of bronchitis in a special degree; such are Bright's disease, gout, diabetes, enteric fever, and, particularly, measles and rickets. (vii.) Heart disease is a potent factor, more especially those forms of it which lead to pulmonary and bronchial congestion. (viii.) Pre-existing chest affections—thoracic, pleural, and pulmonary—also dispose to bronchitis; but none in so well marked a degree as emphysema. (ix.) Relative impurity of air renders the inhabitants of large towns more liable to bronchitis than country folk; but the deprivation of an open-air life, and long sedentary hours in crowded dwellings, are probably more potent influences; and those whose lives are chiefly spent out of doors, even if they perpetually breathe town air, probably do not suffer in the same degree. Dr. Frederick Roberts, in Reynolds' *System of Medicine*, states that in Cheshire and Lancashire, during the year 1868, the ratio of mortality from bronchitis to the number of inhabitants was 1 in 379·5; whilst in London it was only 1 in 442·3. It is suggested that this striking mortality is due to the sedentary lives led by so many mill-hands, to the high temperature of the factories, and perhaps to the effluvia which pervade the manufacturing districts. (x.) The climate of this country, by its humidity and variability, favours the prevalence of bronchitis. Variations occur in the mortality year by year as the weather oscillates more or less. In 1867 it reached 1902 to every million living; but the mean rate for fifteen years, from 1850 to 1864, was 1344·4.

Although sudden changes to cold winds, and particularly to the north-easterly winds, are marked by a large increase of bronchitis, it does not appear that mere bleakness or habitual exposure to strong winds, particularly to the north and to the east winds, so largely tend to set up bronchitis as might be supposed. This is shown, in the figures for 1868, in the relatively favourable return from the eastern counties, which head the list with a mortality of 1 in 987·5 inhabitants, and of the north midland counties (1 in 876·2), as against the south-western counties (1 in 844·8), and all the other districts which have yet higher rates of mortality. We notice, however, that Monmouthshire and Wales (1 in 955·4) closely approach the position taken by the eastern counties.

The difference between the seasons is that which might be expected. Bronchitis is greatly more prevalent during the winter months than in summer, and the liability to it extends into early spring. Thus, whereas the greatest prevalence of pneumonia occurs during March and April, that of bronchitis belongs to the colder months.

(xi.) Aerial impurities may be solid, fluid, or gaseous. Strongly irritating particles or vapours may act as direct exciters of bronchitis, as for instance the vapour of ammonia, of iodine, of bromine; finely powdered ipecacuanha, pepper, or tobacco; and, in the case of those specially liable, the pollen of certain varieties of flowering grass.

B. *Immediate causes.*—The most usual proximate cause is a chill. The patient is said to have "caught cold." The precise meaning of this phrase is obscure. So long as the adaptive mechanisms are in full efficiency, mere extremes of temperature do not constitute a danger to the mucous membrane, and a strong man may pass unscathed from one extreme to the other. Even infants and old people may breathe cold air with impunity, especially if it be dry, so long as they are adequately clad and in perfect health. The liability to "catch cold" is sometimes an individual peculiarity; more often it is acquired, but it is usually intensified by sundry debilitating causes and by faulty hygiene.

Very little is known concerning any functions of the aerial mucous membrane analogous to the regulating mechanisms of the skin for temperature. Their existence is rendered probable not only by the noticeable differences in individual susceptibility, but by the interdependence of the cutaneous and of the bronchial system in the process of "chill." There are two kinds of chill—that directly applied to the air-passages by cold and damp air, the body being at the time warm and well covered; and that which is due mainly to exposure of the cutaneous surface. In both cases the sensation experienced at the time is apt to be referred partly to the skin, the patient "feeling chilly all over," and partly to the air-passages; often to the pharynx or down the trachea. A nervous link is indicated by these paired sensations. Rossbach's experiments show that application of cold to the skin is followed in one or two minutes by a reflex contraction of the tracheal vessels, and a little later by venous congestion and an increased flow of mucus. Any fault in the regulating mechanism, and particularly in its nervous factor, would leave the mucous membrane unprotected against the physical results of continued exposure to extremes; or incapable of that rapid adaptation which is our safeguard against sudden transition from one extreme to another.

Smoke is a powerful irritant, whether by its scorching effect when inhaled hot, by the mechanical action of the suspended carbon or ash, or by the irritating nature of the volatile products of combustion.

Steam, when inhaled from the spout of a kettle by the children of the poor, usually checks inspiration, and its irritating effects are limited to the upper air-passages; but when there is no escape from the inhalation the damage to the air-tubes may be extensive.

Suspended cold moisture, as in ordinary mist, seems capable of irritating very sensitive bronchi, but it is difficult to eliminate the chilling effect of the mist on the body surface; and it is noteworthy that when an equivalent amount of moisture is inhaled in crystalline form, as in a severe frost, its mere cooling effect is not as a rule resented. The nasal passages, of course, exercise some warming influence.

Town fogs are directly responsible for a great deal of bronchitis. Consisting as they do of a mixture of suspended moisture with varying proportions of the products of combustion, fogs differ greatly in their irritating qualities. The fog is acid, and each droplet of water is coated not only with a minute proportion of some tar-like body, but with an equally minute quantity of sulphuric acid; a combination most likely to excite inflammation of the respiratory passages in delicate persons.

Irritant gases have been classified as non-respirable and respirable. To the first group belong chlorine, ammonia, sulphurous anhydride, and the vapours of iodine and bromine. The danger of their continuous inhalation is obviated by the intensity of the irritation causing spasmodic arrest of respiration. A single whiff of ammonia is commonly followed by a transient watery flow from the mucous membrane.

Among the mildly irritating vapours ether, so largely used for surgical purposes, deserves special mention. In the case of small children, in the aged, and in those with limited respiratory surface, its use is to be avoided; even though a proportion of the instances of so-called "ether bronchitis" may be regarded as due to exposure of the surface during the operation, or to the cold produced by the evaporation of the ether, rather than to any direct irritation of the membrane.

As regards temperature, we know that standing in a cold draught, staying out at sunset with insufficient wraps, keeping on wet clothing after severe fatigue, or sitting long with wet or cold feet are so many modes of causation of bronchitis by cutaneous chill. When the impression of chill is confined to the mucous membrane itself, the mischief is usually due less to the intensity of the cold than to previous exposure of the membrane to hot and impure air.

Intolerance of any but the milder kinds of atmosphere is most commonly the artificial result of injudicious physical education. It also belongs to states of debility and to the extremes of age.

The popular belief in the contagious character of common catarrh has received from time to time considerable support from the prevalence of epidemic catarrh and influenza. Although the latter disease does not exclusively attack the respiratory passages, still the almost universal coincidence with it of more or less inflammation of the air-passages must give it a place among the causes of bronchitis. In many instances the irritation, whatever be its mechanism, is severe, the cough being of a harassing type which resembles that due to mechanical irritants, and not infrequently inveterate. Ordinary bronchitis has never been attributed to a specific contagium, so far as I am aware.

Lastly, the possibility of a penetration, through any mucous abrasions, of the micro-organisms of disease, and particularly of the staphylococcus pyogenes, of the streptococcus pyogenes, or of the pneumococcus—not to mention numerous less harmful microbes found in normal air-passages (8 different streptococci, 21 bacilli, 10 micrococci, and several sarcinæ, according to Panzieri)—should not be forgotten. Bronchitis might, it has been suggested, be brought about by a combination of influences, the micro-organisms finding access through an epithelial layer previously loosened or damaged by undue exposure.

Pathological anatomy.—Professor Hamilton's researches,[1] from which the present account is largely derived, furnish us with the most recent data.

(i.) *Acute catarrhal bronchitis* begins with a relaxation and distension of the blood-vessels of the inner fibrous coat; a few hours after this the basement membrane becomes œdematous, much swollen, and folded: twenty to thirty hours afterwards it loses its ciliated cells in patches, and some of these may be inhaled into the smaller bronchial tubes. Immature cells are supplied in great number by the proliferation of Debove's cells, and they constitute the cellular element of the bronchitic secretion. Absolute denudation of the basement membrane may occur, but only temporarily, and over limited areas. Desquamation and active secretion of mucus take place at the same time in the mucous glands. The entire thickness of the bronchial wall is swollen, congested, and infiltrated with leucocytes. Reparative changes are initiated by a diminution in the congestion, and in the dilatation of the vessels; and the cells gradually resume their normal development and functions. Throughout the attack the normal gray colour of the mucosa is replaced by a dull red.

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[1] Cf. *loc. cit.*

(ii.) *Chronic bronchitis.*—(*a*) The common form, the result of a series of acute attacks, is usually associated with much permanent emphysema with intervening congested areas. The lower part of the trachea and the bronchial surface in general are congested and purple, and yellowish muco-pus can be squeezed out of the middle-sized and small air-tubes.

The characteristic smooth and shiny aspect of the mucosa is due to the basement membrane being laid bare, only a few ill-shapen cells adhering to it; it is not always much swollen. Some dilatations may occur in the smaller tubes; the larger ones on the contrary may be slightly narrowed by the great thickening of their coats. The coats are densely infiltrated with cells, among which are seen many dilated capillaries—some of which may project into the thickness of the basement membrane—many thickened arterioles, and over-distended lymphatics; these are especially abundant close to the cartilages which are vacuolated, and in various stages of absorption. The muscular coat may be hypertrophied, or on the contrary greatly atrophied; or even absent. The mucous glands also may be destroyed by cell infiltration, or on the other hand much enlarged, with active mucous transformation of the glandular and duct cells. Atheroma is frequently observed in the middle-sized pulmonary arteries in the subjects of chronic bronchitis.

(*b*) A separate form of chronic bronchitis is characterised by a peribronchitis fibrosa chronica (Virchow), and in some cases the fibrosis extends along the pulmonary lymphatics to the entire interlobular stroma. Instead of the common atrophic, rarefying emphysema, the lung tissue then presents diffuse condensing fibrotic changes.

Physical signs.—The physical signs common to all forms of bronchitis are so familiar that little more than a cursory review of them is necessary.

In shape and in size the chest tends to assume the inspiratory type, without deformity, but with marked elevation of the clavicles and shoulders, deepening the suprasternal and supraclavicular fossæ. In consequence of this and of the hyperinflation of the lungs, the diaphragm, liver, and heart are more or less depressed.

The thorax moves at an increased rate, but to a diminished extent. In severe bronchitis the inspiratory efforts fail to expand the chest, except in its upper part; and there may be inspiratory recession of the lower interspaces, and in children of the lower ribs and sternum. The abdominal muscles are thrown into strong and prolonged contraction during expiration.

Bronchial fremitus is felt on *palpation* during the entire respiratory act, or may be confined to inspiration or to expiration. Vocal and tussive fremitus are not materially altered.

Percussion in most cases elicits an increased resonance, which may, however, be masked by the strong contraction of the inspiratory muscles, to which is also due the peculiar tenderness of the chest. In small children the occurrence of broncho-pneumonia or of collapse may detract from the resonance, or may even cause an imperfect dulness.

Auscultation.—Except at the upper part of the thorax, where they are often exaggerated, the respiratory sounds are much diminished or may be inaudible. Their coarse and harsh character is indirectly due to the feebleness of the alveolar murmur, which no longer veils the sounds produced in the bronchioles; a condition also observed in emphysema.

The adventitious sounds arising in the chest in the course of a simple bronchitis include the two great classes of the dry and of the moist sounds. To the first belong the large or sonorous, the small or sibilant, and the intermediate or subsibilant rhonchi. The Æolian harmony often audible seems to be specially frequent where some emphysema is kept up under the joint influence of bronchitis and of muscular spasm. Another musical sound is the rhythmic sibilus which may be set up in the neighbourhood of the heart by each cardiac systole. Considerable extension and loudness of the bronchitic sounds, although indicating the implication of rather small tubes, and compatible with severe symptoms, are not usually of anxious import in themselves, since they indicate that air passes, though not freely, through a large number of tubes. Clicks are sounds of sudden and snapping character, lacking musical quality and difficult to interpret; being occasionally suggestive of a parched, at other times of a moist, condition of the tubes. Hence they are described in different instances as moist clicks or as dry clicks, thus occupying an intermediate position between the rhonchi and the mucous râles. The moist sounds of bronchitis have a gurgling or bubbling quality. Nomenclature and description are much simplified by calling them *mucous râles* (large, medium-sized, or small), in contrast with the hard or metallic rattles, crackles, or crepitations which may occur in the same chest if broncho-pneumonia, or lobar pneumonia, or phthisis should complicate the bronchial catarrh. The fine crepitations which may become audible over limited patches in capillary bronchitis, in association with abundant mucous râles elsewhere, illustrate this distinction.

Cardiac signs.—In fully established bronchitis a more or less distinct epigastric beat is felt, the heart being not only depressed but enlarged also. The enlargement is mainly due to an over-filling of the right auricle and ventricle, evidenced by the distended jugulars; whilst the left ventricle presents little change. At the same time the absolute dulness of the heart is lessened in its size, and the heart sounds in their loudness by the inflation and encroachment of the sternal fringes of the lung. A relative increase in the loudness of the second pulmonary sound also belongs to uncomplicated bronchitis.

A. SIMPLE BRONCHITIS LIMITED TO THE LARGE AND MIDDLE-SIZED TUBES

Symptoms and course.—At the onset the attack may make itself felt as a severe cold in the chest, with deep-seated rawness, soreness, and parching; or it may begin in the larynx, or in the pharyngeal or nasal region, implicating also the ocular conjunctivæ, the frontal sinuses, and the upper nasal cavities. Again, there may be more or less gastric and hepatic disturbance. Individual peculiarity and local susceptibility may help in each case to determine the site of invasion. That these are not, however, the only factors is shown by the regularity with which special forms, such as the bronchitis of measles, begin in special situations.

In acute cases much continued or intermittent chilliness, and in children slight delirium, or even convulsions (especially during the first dentition), may open the scene.

With every variety of onset there is a uniformity in the general symptoms. The pulse and respiration are moderately quickened, and the temperature is raised two or three degrees. The patient complains of respiratory discomfort, malaise, aching pains, headache, mental and physical languor, drowsiness during waking hours, and restless sleep—the results of the sudden check to the secreting and exhaling functions of a large section of the respiratory membrane. Almost invariably the alimentary mucous membrane is involved: the appetite fails, the tongue is heavily coated, the liver inactive, and the bowels torpid.

The symptoms of the disease when in progress may be classed as general, local, and respiratory. The local pain is seldom acute. The sensation is almost always retrosternal; it is variously described as "sore," "raw," or "burning," and the cough as "tearing." Tenderness on pressure is also felt at the sternum, but greater tenderness arises later from the constant strain of cough, and is then felt over the entire chest, but particularly over the pectoral muscles and at the base of the thorax.

The general symptoms are those of slight feverishness. The dry heat of the skin which follows the stage of invasion in most cases soon gives way to moisture. The temperature oscillates in the usual manner between a morning minimum and a maximum at night, but does not often rise very high. The pulse is moderately quickened and full; at first it is excited in action and almost bounding, but subsequently, with the advent of diaphoresis, large, soft, and undulating. The tongue is furred but moist, and the appetite bad; vomiting is unusual, constipation almost the rule. The urine is of the febrile type, with rather high specific gravity; in healthy subjects it is free from albumin, but loaded with lithates, pigment, and urea; sometimes it contains less than the usual amount of sodium chloride.

Respiratory symptoms.—The patient's complaint is of tightness and oppression at the chest, rather than of dyspnœa, though this would be brought out on any exertion. Even in the position of rest the respirations are markedly quickened and proportionately more so than the pulse; they are shallow, and ultimately become laboured. Cough sets in early, especially in the laryngeal, tracheal, and bronchial forms of onset; rather later when the pharynx is affected first, and sometimes not for a day or two when the attack begins with coryza, in which cases sneezing is more common. At first the cough is dry and irritating, and usually associated with a tickling sensation in the larynx or trachea; when these structures are involved it is much altered in tone. It is easily set up by slight irritation, and is difficult to check. At a later date the paroxysmal character is no longer due to simple irritation of the nerve-endings over a dry and parched surface, or to a congested uvula and epiglottis, but to the difficulty in expelling the viscid and frothy secretion.

The expectoration, in all cases of bronchitis, furnishes us with indications as to the stage and progress of the affection. From the healthy state of simple moisture, free from any perceptible excess of fluid or of mucus, the inflamed membrane, after a preliminary phase of checked secretion and of dryness, quickly passes through a stage of excessive hydration, during which the mucin of the cells is matured; though but little of it finds its way into the saline watery flux. After a very few hours mucus is poured out more freely, and renders the fluid ropy; but it is still as transparent as glass, and free from bubbles other than those produced in the mouth or in the larger tubes. The next stage is that of purely mucous catarrh. The secretion stiffens, and, in the smaller tubes, soon offers considerable resistance to the respiratory current. This is clearly seen in the amount of air-bubbles held in the mucus, which, although in itself hyaline and colourless, forms with them a white opaque froth. This is the "crude stage" described in ancient books. In cases of rapid resolution the mucus may soon undergo a secondary hydration, losing its bubbles, and coming up with less effort and in rapidly lessening quantities.

More commonly in the ordinary case of bronchitis the sputum passes through some degree of "coction" (to use again an obsolete term), losing together with its extreme viscidity and frothiness the hyaline colourless quality, and becoming either streaked or uniformly tinged with light yellow pus, whilst continuing to form as before a confluent mass in the receiver. In more protracted cases the admixture of pus gradually increases, and imparts a greater opacity and a greenish tinge to the sputum, which becomes less hydrated, quite free from bubbles, and ultimately nummular. This is a sign that the catarrhal process is lingering in the larger tubes. There is much analogy and yet a distinction between this expectoration and the more purulent and fluid discharge which from its quantity and inveterate character has received the name of purulent bronchorrhœa, and in which the individual sputa fuse into a mawkish yellowish semi-fluid mass. In the later stage of bronchitis the sputa remain distinct.

Hæmoptysis, in simple uncomplicated bronchitis, is of exceptional occurrence; but a few streaks of blood may be seen in the earlier and drier stage. They are probably due to the sudden detachment of superficial layers of the membrane under the effort of cough.

Prognosis.—As to the duration of the attack prognosis is of necessity somewhat uncertain, and is partly governed by atmospheric conditions. In healthy children, youths, and adults, especially if not previously affected, complete recovery under appropriate treatment may be looked for within one or two weeks, according to the severity and extent of the inflammation. Any antecedent bronchial trouble would modify and unsettle the estimate. In infants and the aged it is wise not to fix any date.

As to danger to life, it is only at the two extremes of age, and in albuminuria, or diabetes, or heart disease, or cachexia, that doubt is likely to arise. However much they may ultimately tend to shorten life, even repeated attacks of this mild form of bronchitis are never directly fatal in subjects otherwise sound. If the respiratory muscles be feeble, as in infancy, old age, or obesity, there is risk of *broncho-pneumonia a retentis*, the termination of which cannot be foretold; the other risk, peculiar to the same group of patients, arises from weakness of the heart, and especially of the right heart, which may undergo dilatation and eventually paralysis; or the bronchitis, especially in the aged, may become chronic, and prove at length a fatal drain on an exhausted vitality.

B. ACUTE SUFFOCATIVE BRONCHITIS OF ADULTS, OR BRONCHITIS OF THE SMALL TUBES

Symptoms.—The following sketch of the clinical history of suffocative bronchitis may justify our attempt to deal with it as a separate study. Walshe, who obviously appreciated the essential differences between it and capillary bronchitis, nevertheless included their description under one heading,—"General and capillary bronchitis—olim peripneumonia notha"; and subsequent authors have followed him.

A first distinctive feature of simple asphyxial bronchitis is the exceedingly rapid and general implication of the small tubes throughout the lung. Walshe says: "I have known life destroyed in forty-six hours, reckoning from the first moment of seizure, in a middle-aged adult, who, in previous years, had had more than one seizure." In the adult (and it is noteworthy that young adults are rarely attacked) orthopnœa is the rule, and, as observed by Walshe, "Maintenance of the head on a low level from the first, in a case otherwise grave, is of evil augury." It is hard to say to what extent superadded muscular spasm of the bronchioles may increase the constriction due to inflammatory swelling.

More air is at first drawn into the lung by the strenuous breathing than can be expelled by expiration. Subsequently, in spite of the powerful contractions of the muscles of extraordinary respiration, the chest moves comparatively little, and ultimately the character of the respiration tends more and more to become expiratory and abdominal. The lower intercostal spaces are drawn in with each inspiration, but the ribs do not usually recede. The whole chest is enlarged, and the lungs over-distended by the powerful muscular forces applied to sufficiently rigid bones and cartilages. An excess of air is, as it were, locked in by the obstruction of the bronchioles: henceforth little passes through them into the lobules, whether in the shape of air or of secretion; and the direction taken by the latter is outwards, not inwards as in capillary bronchitis. The oxygen of the imprisoned air becomes exhausted, and the turgid veins and the asphyxial complexion of the patient warn us of the degree of the obstruction to the pulmonary circulation, and of the congestion of the overloaded right heart.

Expectoration is not suppressed as often occurs in capillary bronchitis. A fine white foam resembling "whipped egg" gives in the minute size of its bubbles the gauge of the tubes affected. An analogous "whipped egg" sputum (not, however, quite so fine) is sometimes observed in the sudden pulmonary congestion apt to complicate an anginal attack. In the absence of angina this sputum is diagnostic of suffocative bronchitis. A change to a coarser froth with the admixture of watery, hyaline, and subsequently of purulent mucus gradually occurs in the later stages of the more favourable cases.

Asphyxiating bronchitis of the adult is not complicated with any parenchymatous inflammation of the lung. Pneumonia is perhaps mechanically obviated by the intra-alveolar pressure of gas, and by the stretching of the alveolar vessels. At any rate this immunity is attested by the pulmonary appearances after death and by the observations of every clinical observer. Walshe says: "True pneumonia, lobular or diffused, is of purely exceptional occurrence; the parenchyma is often even unusually pale"; and again, "but, without meaning to deny the possibility of the fact, I must observe I have never yet seen local collapse of lobules on an extensive enough scale in simple adult bronchitis (antagonised as it is by the distending influence of the disease on the alveoli) to justify me in looking upon it as a sufficing cause of deficiency of tone"; once more he says, "Bleeding is useless for the prevention of pneumonia, seeing that, in the adult, idiopathic inflammation of the tubes does not pass on to the parenchyma."

The later course of the disease need not be detailed at full length. The symptoms are those of a progressive asphyxia—a prolonged struggle for breath, the duration of which is measured by the patient's cardiac energy. In Walshe's unsurpassed description:—

"As long as his strength permits, the patient sits erect or bends forward; but the body gradually yields; and it is not uncommon to find patients, while still perfectly conscious, lying sidewards or forwards with the head lower than the shoulders. In rare cases, a posture of this kind is adopted from the very onset.

"The sputa gradually diminish in quantity from failure of power to expectorate; the skin, generally livid or cyanotic in tint, falls in temperature, becomes covered with cold, clammy perspiration — sometimes copious, rarely attended with formation of sudamina; the expired air grows cool, the feet and hands swell, in protracted cases the anasarca rising to the trunk, unaided by coexistent disease of the heart, or of any other organ promotive of dropsy; fitful dozes lapse into a state of somnolescence, constant, except from momentary interruptions by the cough; muttering delirium, associated in some instances with slight convulsions, precedes a comatose state which is the immediate forerunner of death."

The pulse gains in frequency as it loses in power, ranging from 120 to 150 or more. The respirations, varying from 36 to 50, may ultimately recede from the maximum rate they had attained.

The temperature is moderately elevated. Dyspnœa, oppression, retrosternal pain, restlessness, and cough are the chief symptoms complained of.

The urine is scanty and concentrated. There is occasionally a transient albuminuria, but, in spite of the great diminution in the oxygen supply, there is no sugar, and usually no excess of urates.

The physical signs are those of emphysema, as regards increased bulk of the chest and of the lung, depression of the diaphragm and of the heart, and pulmonary hyperresonance, coupled with bronchitic râles in the larger tubes owing to the ascent within them of the frothy secretion.

The prognosis is anxious even in the best subjects. The worst cases are those of pre-existing emphysema with incipient or advanced dilatation of the right heart; these subjects seldom long survive the onset of a genuine bronchitis of the small tubes. Cardiac defects, or inherent debility, whether from exhaustion or atheroma, chronic albuminuria and the various cachexies greatly reduce the chances of recovery.

The duration of a fatal attack may be reckoned in hours, or may "drag on to the tenth or twelfth day" (Walshe). The same authority has recorded unexpected recovery after long periods of an apparently hopeless condition, with cold clammy sweat and almost complete loss of conjunctival reflex. Such cases are rare; they seem to suggest that spasm of the bronchioles had contributed to the bronchial stoppage.

Morbid anatomy.—The post-mortem appearances are almost invariably those of an over-distended, non-collapsing lung, the pale pink colour of which contrasts strangely with the deep cyanosis of the body surface, and is readily explained by the influence of the residual oxygen of the distended air-cells on the reduced quantity of blood which their overstretched capillaries accommodate. The small bronchi present, on the contrary, a swollen and deep red surface of section. Their contents vary with the duration of the cases: in early deaths they consist chiefly of mucus; they are semi-purulent in those who have survived for several days. Exceptionally here and there a pulmonary lobule may be found collapsed, but pneumonic consolidation is absent. In all cases the heart presents the asphyxial condition, and the viscera are engorged.

C. CAPILLARY BRONCHITIS OF INFANCY AND OLD AGE (PERIPNEUMONIA NOTHA)

This name specially belongs to the inflammation which extends from the small bronchial tubes into the lobules and into the alveoli. It is in great part a pneumonia, and was originally observed and referred to as such long before the existence of bronchitis as a disease was thought of. Indeed the pneumonic changes, where they coexist with bronchitis, are obvious enough; whilst of all diseases, equally intense and clinically definite, none leaves after death traces slighter in themselves, or more easily overlooked by the inexperienced observer, than those of simple acute bronchitis.

It is singular that this liability to the pneumonic complications should be shared exclusively by the earliest and by the latest stages of life, in spite of most opposite anatomical conditions; the lung being atrophied and rarefied in old age, with relatively large air-spaces and tubes, whilst in the infant it is only partly developed, and is fully packed with relatively narrow air-tubes and air-cells yet imperfectly expanded: the thorax in the one case is roomy and almost rigid, in the other relatively smaller than at a later date, and exceedingly yielding; the morbid processes reflecting in the first the sluggishness of age, in the second the activity of budding life. It can hardly be doubted that the intimate changes in the two conditions must present essential differences, and that the similarity between them must reside mainly in the general lines of march of the disease, and in the direction taken by its extension. At any rate in the capillary bronchitis of infants we perceive a factor entirely foreign to that of old age, the tendency to rapid proliferation of the tissue elements under irritation, and to the choking of space by direct cell overgrowth. One peculiarity is common to both extremes of life—feebleness of the mechanisms of respiration, which allows the obstruction to tell in a degree not witnessed in the adult. In other respects the processes differ.

Infantile bronchitis attacking the small tubes almost inevitably disables some of them at an early date, owing to the very unequal local resistances of the chest walls, and to the influence of decubitus. At given spots the thorax fails to draw out the subjacent lung, and is dragged in instead. The subjacent lobules quickly become airless and collapsed, and are henceforth sealed against the entrance of gases, fluids, and solids alike; they are incapable of becoming pneumonic. Collateral emphysema results from the increased respiratory stress thrown on other parts, and, thanks to their early over-distension, these lobules also may remain free from pneumonia. It is in the remaining portions of the lung, imperfectly expanded and traversed by enfeebled respiratory currents, that the changes occur. The secretion, failing to be expelled by its stagnation, sets up intralobular irritation, and a tissue-reaction which is mainly proliferative.

Two forms are noticed — the strictly broncho-pneumonic with prevailing proliferation of epithelia, infiltration of the bronchial and alveolar walls, and consolidation of the alveoli by epithelial cells; and the purulent form in which loose, semi-fluid bronchial secretion accumulates in the smaller divisions, dilating many of them and setting up a form of acute generalised bronchiectasis;[1] whilst a varying amount of pneumonic change is also present.

In old age rigidity of the thorax, degenerative changes in the lung, such as widening of the alveoli and of the air-tubes, atheroma of the pulmonary artery and relaxation of the pulmonary veins, loss of inspiratory energy and considerable loss of general expiratory power, and especially of the expulsive power of individual districts the expansibility of which may have been reduced by pleural adhesions or by the reticular fibrosis left behind by former attacks, are some of the factors determining the variety of capillary bronchitis. The minute diameters of the tubes and the yielding of the thoracic parietes, to which are due the pulmonary collapse and collateral emphysema distinctive of the infantile form, are conditions conspicuously absent. The character is that of passive retention rather than of primary bronchial obstruction, though this element is not entirely excluded. Gravitation has a larger share in determining the locality of the changes; and the basic and posterior regions are affected with much greater regularity than in the infant. For these and other reasons the ingravescent, slowly developing form, beginning in the medium-sized tubes, is of special frequency in senile bronchitis. Again, the tissue reaction is of a different quality. Peribronchitis and alveolar wall infiltrations are ill-developed. The consolidations are more definitely broncho-pneumonic or terminal, and, owing to the even operation of gravitation, tend to be confluent. At the same time they are less dense and are usually combined with passive congestion and with œdema, which are not features of the infantile variety.

The preceding remarks must have made it clear that between capillary bronchitis and broncho-pneumonia it is difficult to draw a hard and fast line: the one and the other are made up of bronchitis and of pneumonia. Nevertheless, on clinical as well as on pathological grounds, it is desirable to uphold both forms in our nomenclature. Capillary bronchitis, as bronchitis, is always a general affection of the entire lung leading to severe dyspnœa. It is quite true than broncho-pneumonia in its worst forms often becomes generalised and leads to intense breathlessness, but these are results which need time for their manifestation; their evolution is comparatively gradual, in opposition to the early and often rapid onset of the dyspnœa of capillary bronchitis. Again, although capillary bronchitis, especially in infants, always tends to set up some parenchymatous inflammation, the occurrence of a broncho-pneumonia is not invariable, death may occur from the bronchitis before time is given for the consolidation; and in other cases the tendency is rather to peribronchitis, to purulent infiltration of the bronchi, and to dilatation, than to consolidation.

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[1] For two interesting cases of this kind, and for excellent drawings of sections of the lungs, see a paper by Dr. Sharkey in *St. Thomas's Hospital Reports*, 1894, and the writer's article on "Bronchiectasis" in the present volume. Bronchiolectasis has also been suggested by Dr. Tooth and Dr. T. H. Fisher. *Vide* Dr. Tooth, *Path. Soc. Trans.* vol. xlviii. pp. 30-34.

Symptoms and Diagnosis.—In the infant or young child the history of the attack, combined with the physical signs about to be described, generally suffices to establish the diagnosis.

The signs are those of pulmonary collapse at the anterior and lateral base of the thorax with inspiratory inward suction of the costal arch, and of emphysema of the upper part of the lung. The resonance due to the latter disguises the dulness which otherwise might have arisen from any pneumonic condensation. Nevertheless examination may reveal a lack of freedom and fulness of the auditory and tactile vibrations. Little air enters the chest in spite of the strenuous efforts of the upper inspiratory muscles and of the diaphragm, the contractions of which drag the sides of the chest inwards instead of expanding them. Sibili may be heard at first, but they are soon replaced or silenced by râles, the loudness of which, always great in the small chests of children, is intensified by any existing collapse or consolidation, and precludes the distinct perception of any bronchiolar or tubular breath sound. In reality these râles, the only sounds audible, do not arise in the capillary bronchi, but are produced by the to-and-fro movements of the secretion within the imperfectly swept medium-sized and larger tubes.

Exhaustion is an early feature; the patients, if not too young to be able to sit up in orthopnœa, rapidly lose that power; and lie pale, livid, and helpless, with hurried respiration, distended nostrils, and extremely rapid pulse. Expectoration does not occur in the younger children, or but rarely, and from an early period in the disease cough may be absent; but both the cough and the dyspnœa are prone to paroxysmal aggravations after remissions.

The temperature varies with the amount of pneumonic action, but probably also with the susceptibility of the individual nervous system. It may rapidly lessen with the advent of cardiac exhaustion and coma.

The disease is usually fatal, and the **prognosis**, except in relatively robust constitutions, is practically hopeless. The duration of the urgent symptoms varies, but, for obvious reasons, is on the average much shorter than in ordinary broncho-pneumonia. The acute stage of the disease does not often exceed five or six days; it commonly destroys life at an earlier date.

In the aged the affection is usually ushered in by a pharyngeal, tracheal, or bronchial cold, which more or less gradually assumes the character of general bronchitis; or it may be grafted upon a chronic catarrh. The extension of the inflammation to the bronchioles is marked by moderate pyrexia, paroxysmal cough and dyspnœa, laboured expectoration, a dusky flush changing to pallor, a rise in the rate of pulse and respiration, and great prostration. All appetite is lost, the tongue becomes dry and brown, and muttering delirium sets in, to be followed by deepening coma. In the less rapid cases, evidence of a low form of broncho-pneumonia, associated with œdema and with the signs of bronchitis, may be ultimately obtained at the bases; but, as a rule, the exhausted state of the patient forbids any searching examination of the posterior pulmonary regions. In extreme old age treatment is unavailing, and the disease is almost invariably fatal.

D. CHRONIC BRONCHITIS, CHRONIC BRONCHIAL CATARRH, AND BRONCHORRHŒA

In this brief review of a wide and important subject, Walshe's division into four clinical groups will be adopted.

(*a*) *The simple winter cough*, moderate, not disabling, accompanied with the easy expectoration of a yellowish white muco-pus, is merely an expression of the bronchial irritation set up by atmospheric conditions; it is frequently observed in children and young adults, as well as in older people.

(*b*) An aggravated form of the same winter cough is peculiar to *chronic bronchial catarrh.* The health and strength suffer; and the patients are invalids, though often struggling to pursue their avocations. Decided functional and some organic change may be traced in the organs of respiration, of circulation, and of alimentation; such as short breath, venous fulness both general and portal, and delicate digestion. The winter is spent in a succession of slight pyrexial relapses, during which the expectoration, habitually loose and muco-purulent, may, after being frothy for a while and difficult to raise, become unduly abundant and puriform. The feverish bouts may last a week or a fortnight, during which the appetite is in abeyance, the tongue, stomach, and liver are out of order, and considerable weight is lost. Between these attacks the patient regains some of the previous health and strength, but never shakes off the cough, which may even last, in a modified degree, through the summer.

These patients are protected from graver risks by their general delicacy and invalidism; but the process is progressive and devitalising through the inevitable changes it causes in the lung and in the heart. It induces premature senility and shortens life.

(*c*) *Bronchorrhœa* indicates a special group in which the constitutional factor has probably no less a share than the pulmonary changes. Two varieties need description:—

(i.) The thin mucous or thin watery bronchorrhœa is thus described by Walshe:—"In this variety paroxysms of cough and dyspnœa, which may be of almost daily occurrence, or even more frequent, are relieved by copious expectoration of a thin, watery fluid, or of a ropy, gluey, transparent substance, like raw white of egg mixed with water; a quarter of a pint of this may be secreted in the course of half an hour on the decline of a paroxysm." Though sometimes fatal in the aged, the flux is regarded by Walshe as occasionally useful in relieving pulmonary congestion due to mitral disease. This singular affection is well identified by the name of "bronchorrhœa serosa" given to it by Biermer, by that of "mucoid asthma," or by its original name "chronic pituitous catarrh," used by Laennec. The paroxysms of dyspnœa and mucorrhœa may be of isolated occurrence in the morning after waking; and the chest, after two hours, may be comparatively clear for the day: or the discharge may be repeated once or twice, producing in extreme instances a daily output of three or four pints; and this may last for years (Laennec). Lebert mentions a case of survival to the age of eighty-two, after thirty years of bronchorrhœa; but Wilson Fox regards gradual failure as being the common tendency, together with increasing dyspnœa and delicacy of digestion. Pulmonary and cardiac degeneracy progressively lead to emaciation, anæmia, cyanosis, œdema, and exhaustion.

Much obscurity still attaches to the pathology of the affection; and it is still doubtful whether the disease is primarily associated with emphysema and bronchiectasis, or whether these be merely secondary changes.

(ii.) Purulent bronchorrhœa or bronchial catarrh is a severe, inveterate, and progressive affection refractory to all treatment except the climatic. The bronchial discharge resembles in general character that observed in the diffluent stage of chronic catarrhal bronchitis during the exacerbations noted under (*b*); but generally exceeds it in quantity, and in the semi-fluid, mucoid nature and mawkish odour of the pus. Pathologically the affection differs from simple chronic catarrh, chiefly in the extent of the bronchial and pulmonary change. The mucous membrane is thickened, the bronchial walls infiltrated, and the calibre of the smaller tubes increased, though there need be no sacculation or extensive cylindrical dilatations such as belong to bronchiectasis. Between these two conditions there is, however, no strict demarcation, and mixed forms are met with. Emphysema is a direct and never-failing result of the loaded state of the bronchial system, and of the constant strain of cough. The atrophy of the pulmonary parenchyma contributes the progressive element in the disease, and renders it intractable after it has lasted for considerable periods.

The amount of the expectoration, which may reach one, two, or even three pints daily, is in itself a serious drain; and the cough is a severe tax on the strength. Night sweats, an habitually subpyrexial temperature, and the recurring anorexia or dyspepsia are additional depressants. In connection with the latter, or with disturbance in the function of the liver, or with temporary retentions within, or inflammatory conditions of some of the bronchi, the mawkish secretion may become fetid in odour, sometimes almost gangrenous; and this reacts most unfavourably on the general state.

In spite of these distressing and wearing symptoms, the resistance of some of the patients to the fatal tendency of the disease is remarkable, and should encourage every effort to procure for them the healing effect of appropriate climate. Failing this, recurring winters bring with them steady aggravation, and life may be cut short by intercurrent bronchopneumonia, or may lapse from gradual exhaustion and cardiac dilatation.

(*d*) In the fourth classical variety, that of *dry chronic bronchitis*, the sputum presents characters exactly opposite to those which have just been described. It is extremely scanty, and consists of semi-transparent, tough, pearl-like, roundish, small masses, apparently a highly concentrated and partly dehydrated form of hyaline mucus, in which Charcot-Leyden crystals or Curschmann's spirals are usually contained. The peculiarity of the sputum led Laennec to apply to the affection the name of "dry catarrh," although at times a little watery fluid may be expectorated. The distinctive clinical features are the distressing paroxysmal cough, causing much soreness at the chest; and the dyspnœa and oppression of breathing, intensified by the cough, but in most cases kept up by the emphysema, which almost invariably complicates these cases. Laennec described this form as exceedingly prevalent; but, as pointed out by Wilson Fox, he included under "dry catarrh" not only the asthmatic cases, but all forms of nervous and sympathetic cough (gastric, hepatic, hysterical). Walshe regarded the symptoms as mainly due to active congestion of the tubes. Bronchial spasm is doubtless largely associated with the congestion. Indeed, bronchial susceptibility and bronchial irritation are its unmistakable etiological factors. One of the forms of chronic gouty bronchitis belongs to this type. Dry catarrh is also said to be prevalent at seaside places, and to occur after the cure of chronic cutaneous eruptions, and in those weakened by excesses (F. Roberts). The physical signs are those of the dry stage of acute bronchitis.

II. The Secondary and the Special Varieties of Bronchitis

A. INTERCURRENT BRONCHITIS

This malady is a complication common to many acute disorders; it will suffice briefly to indicate the relation which the bronchial affection bears to the several diseases.

(i.) The most important group is formed by prevalent affections such as whooping-cough, influenza, summer catarrh, phthisis, and measles; of the last bronchitis is an essential and prominent feature.

(ii.) In some of the continued fevers, but especially in typhoid fever, a varying degree of bronchitis is almost the rule; but its importance is rarely of the first order, and the same remark applies to cases of typhus fever. In enteric fever the severity of the early bronchial catarrh may occasionally mislead the physician for a day or two; and in protracted and exceptional cases the unabated persistence of bronchial râles may arouse uneasy suspicions of general tuberculosis. A malarial bronchitis has also been described.

In scarlet fever and in small-pox bronchitis is not a regular symptom. The occurrence of bronchitis in rheumatic fever, fortunately infrequent, was, before the introduction of the salicylic treatment, a most painful and dreaded complication; it still remains a serious trouble, in spite of the earlier relief afforded to the articular pain.

(iii.) In other affections bronchitis is only an occasional complication. Among them chronic disease of the kidney probably takes the chief place, both as regards the occurrence and the gravity of secondary bronchitis. Gout is also prominent for the frequency of bronchial symptoms. Reference has already been made to the "dry bronchial irritation" so often observed in the gouty, independently of any articular seizure. In cases of retrocedent gout bronchitis may assume alarming severity, and is then probably characterised by extreme congestion. Severe bronchitis of a congestive and catarrhal type may, however, also occur as a precursor of the arthritic attack, usually subsiding with the onset of the latter. A syphilitic bronchitis was described by Graves, by Stokes, and by Munck; and Walshe bestows two pages upon its discussion. It was supposed to occur prior to the cutaneous eruption, and to alternate in gravity with the latter. Bronchitis was also described as complicating cases of syphilis between the secondary and the tertiary stages; and again during the tertiary stage, when it might be unilateral, whereas in the secondary stage it was said to be invariably bilateral. Nothing has been added to Walshe's description, which is reproduced by Wilson Fox. Indeed, syphilitic bronchitis does not now hold any independent place, and of late years has obtained no recognition; although considerable attention has been given meanwhile to the study of pulmonary syphilis (*vide* p. 311). The scrofulous bronchitis of Graves is another constitutional variety which has failed to obtain a permanent footing in the practical nomenclature of the disease. Among blood diseases, anæmia, chlorosis, and pernicious anæmia do not especially favor the occurrence of bronchitis. In scurvy, however, bronchitis is not an infrequent complication, and is often associated with hæmoptysis, which does not belong in a comparable degree to any of the other forms, except the phthisical and the cardiac.

Walshe draws attention to the occasional admixture with the sputum of substances derived from the blood; such as bile in icterus, sugar in glycosuria and diabetes, urea or its products in uræmia.

(iv.) A special group may be made of those forms of bronchitis which are dependent upon pre-existing pulmonary or intrathoracic disease. Aneurysm, mediastinal growths, or cicatricial stricture of a bronchus (usually syphilitic, *vide* pp. 71 and 326) may give rise below the seat of stenosis to a localised bronchitis or bronchiectasis, and this may ultimately lead to disorganisation of the pulmonary substance. This result, which is very apt to follow in the rare instances of primary malignant disease of the bronchial mucous membrane, is not often observed in that of the lung, nor in secondary peribronchial malignant disease, whether generalised or occurring in single or multiple deposits. I have observed that the presence within the lung of separate malignant masses of moderate size, even in large number, may, owing to the distension of the intervening pulmonary tissue, give no signs of consolidation either auscultatory or percussive, and yield only the common physical signs of bronchitis. Gangrenous ulceration into the root of the lung or into a large bronchus is a frequent mode of death in œsophageal cancer, and is preceded by the signs of severe bronchial irritation. Emphysema stands in the most intimate relation to bronchitis, both as cause and effect. This association is fully dealt with in another article (*vide* p. 273). The close connection existing between pleurisy, bronchitis, and catarrh is a matter of every-day clinical observation, and it will be briefly studied under a special heading. Pulmonary phthisis is invariably in part, and often to a great extent, a bronchitic process: it is enough to indicate that, in addition to the general bronchitis which is an intermittent complication of most cases, the local deposits and the local pleurisies of early phthisis determine strictly localised bronchial catarrhs which often raise the first alarm and suggest an examination of the sputum. Lastly, acute pneumonia is sometimes associated with well-marked bronchitis, and forms a most serious, though by no means necessarily fatal, complication. I have observed bronchial hæmorrhage persisting for several days as a result of this combination. In the pneumonia of influenza the association with bronchitis is the rule; but here the relation between the two diseases is reversed. Bronchitis begins and pneumonia may follow. (*Vide* art. "Influenza," vol. i. p. 679.)

(v.) Another special place must be reserved for the truly secondary bronchitis of mitral disease, in which clinically, as well as anatomically, three stages may be indicated: (*a*) A passive congestion of the mucous membrane, the mechanism of which has been described by every writer on valvular disease of the heart as the chief cause of the well-known "heart-cough"—short, slight, dry, and habitual, and especially common in mitral stenosis. (*b*) A mild chronic catarrhal bronchitis, easily set up and difficult to throw off, may occur in both kinds of mitral disease; but is most frequent in mitral regurgitation. It is not, or is but occasionally, associated with streaking of the sputum. (*c*) A disabling acute bronchitis is the almost invariable agent in overthrowing the fine adjustment previously maintained between the task and the strength of the ventricles. The rest and the treatment necessitated by the cardiac breakdown may subdue for a time the bronchial trouble; but in both forms of valvular disease the bronchial complication inevitably reappears with the relapsing failure of energy of the right heart. At this final stage the process is almost entirely passive and dependent upon the engorgement of the bronchial circulation. In cases of pure mitral stenosis previous pulmonary apoplexies may have cleared up; but their aggravated recurrence often has a direct share in hastening the fatal event. More commonly, in mitral stenosis combined with regurgitation as well as in pure mitral reflux, the expectoration becomes watery with the onset of hypostatic pulmonary congestion and œdema; and the final obstruction of the air-tubes with frothy mucus is the immediate result of cardiac and of general failure.

B. MECHANICAL BRONCHITIS

Acute mechanical bronchitis. — Hay asthma is the most striking example of the production of acute symptoms from the mechanical irritation of suspended particles. The stronger irritants, such as the sternutatories, cannot be long tolerated, and their effect is momentary and slight. No such safeguard limits the inhalation of the pollen of *Anthoxanthum odoratum*, so noxious to a small class of sufferers. The irritation may involve the entire respiratory tract, including its diverticula, from the frontal sinuses to the small bronchi. Violent and continued sneezing, dyspnœa occurring in paroxysms, oppression and substernal soreness, and wearisome cough, which is at first dry but ultimately may produce a varying amount of watery, mucoid, or faintly opaque expectoration, are the main symptoms in cases involving the bronchi. For a further account of this disease the reader is referred to the article on Asthma in the present volume (p. 286).

Chronic mechanical bronchitis is the main clinical feature and the pathological starting-point of all pulmonary diseases due to the inhalation of dust; whether this be vegetable, as in the case of unloaders of grain-ships, grinders of cereals, hemp-spinners, cotton-batters, and coal-miners; or animal, as in that of wool-carders, bedding-makers, brush-makers, and bristle-drawers; or mineral, as in that of stone-cutters, quarrymen, glass-cutters, and calico-weavers (from the china clay used in calico-making); or lastly metallic, as in that of knife-grinders, metal-turners, and needle-pointers (Walshe). In the early stages of all these varieties the symptoms and the physical signs are exclusively those of bronchitis—the sputum alone yielding on examination the special clue to the nature of the irritant. Sooner or later in all of them the mischief strikes deeper; and to the bronchial catarrh, which becomes permanent, are superadded indurative or destructive parenchymatous changes, causing the affections to be classed under the heading of interstitial pneumonia or of phthisis, under which their description will be found. [*Vide* art. "Pneumoconiosis," p. 242 in the present volume.]

C. PARASITIC AFFECTIONS OF BRONCHI

More closely allied in some of their aspects to mechanical bronchitis than to any other affection are the parasitic pulmonary diseases affecting the bronchi,—detailed descriptions of which belong to other sections of this work; namely, hydatid disease and pulmonary distomiasis in the group of animal parasites (vol. ii. p. 1102), actinomycosis (vol. ii. p. 81) and aspergillosis (p. 257) in the vegetable group.

The *Endemic Parasitic Hœmoptysis* of some parts of Japan, of Corea, and of Formosa was, in 1880, simultaneously and independently traced by Manson and by Balz to its cause, the settling of the *Distoma Ringeri* or

Westmanii near the root of the lung, and the periodical discharge of its yellowish-brown ova into the bronchi. The rusty expectoration resembles that of pneumonia rather than that of bronchitis, whilst the anæmia and progressive wasting are analogous to those of phthisis but have a much more protracted course. Cases of pulmonary distomiasis have not hitherto been reported in this country.

Hydatid disease and the rupture of a hydatid into the bronchi may result in considerable bronchial irritation; but the clinical details of the affection cannot be described here. The occurrence of the *cysticercus* in the lung is exceedingly rare.

Lastly, we owe to Diesing the account of a unique case of the presence of *Strongylus longevaginatus* in the bronchus of a child.

Among the vegetable parasites the most important is the *Actinomyces.* Pulmonary actinomycosis has long been mistaken for the catarrh of phthisis, of bronchiectasis, and of putrid bronchitis. It can now be readily identified by the discovery in the sputum of the clubbed radiating threads of the fungus, which were first described by Bollinger in 1870.

Pulmonary aspergillosis, relatively common in animals and rare in man, still occupies a somewhat doubtful position in pathology. Originally described by Virchow as a separate disease, the invasion of the fungus had since then been regarded as a mere complication of phthisis and of chronic bronchial affections. Latterly the tendency has been to ascribe to the aspergilli, and particularly to the *Aspergillus fumigatus,* primary pathogenetic effects. Rénon, the latest writer on this subject, considers that in some cases the pulmonary and bronchial affections which had been attributed to tubercle or to actinomycosis were really due to the aspergillus (*vide* p. 257).

Glanders.—Although the bacillus mallei, like that of tubercle, is not limited to the lung, it deserves to be mentioned in connection with the bronchial catarrh to which it gives rise (vol. ii. p. 513).

D. BRONCHITIS AND BRONCHIAL CATARRH IN THEIR RELATION TO PLEURISY

(*a*) **Acute pleurisy with bronchitis, or acute pleuro-bronchitis.**—The not infrequent association of acute pleurisy with an acute bronchitis of the middle-sized tubes is the more worthy of attention, as there is not between these affections that necessary nexus which exists between pleurisy and acute pneumonia; and their occasional combination may be regarded as a definite clinical complex. This view finds support in the etiology and mode of onset, the two affections often arising from one and the same exciting cause and with a simultaneous invasion. I have long been in the habit of using the name "pleuro-bronchitis" to suggest something more than an accidental coincidence; some definite tendency in the subject, and some definite relationship between the pathological processes. Rheumatism seems to be the constitutional tendency, and a simultaneous implication of the pleural and bronchial lymphatics the most plausible explanation of the process. The occasional occurrence of bronchitis in conjunction with rheumatic fever makes it the more probable that the rheumatic tendency, in itself so often answerable for attacks of pleurisy, may be at the root of this association, even in the absence of any arthritic manifestations; in the same way as non-articular gout is a common and fully recognised factor in the causation of bronchitis.

Cases of this kind are usually classed as "pleurisies with bronchitis as a complication"—a description justified by the relative prominence of the two sets of symptoms. When the pleurisy, as often happens, is of the dry variety, the physical signs of the bronchitis are those most easily obtained; whilst the most urgent symptoms belong rather to the pleural affection. In cases with considerable effusion this relation is reversed; extensive dulness is a prominent physical sign, but the urgency of the symptoms is largely due to the bronchitis, and is often in excess of the loudness of the auscultatory signs special to the latter. When the diaphragm is implicated in the pleurisy, the combined affection assumes unusually severe features, owing to the acutely painful dyspnœa, and to the interference with the mechanical function of cough in clearing the air-tubes.

(β) **Chronic bronchial catarrh associated with pleuritic adhesions.**—Strictly speaking, the affection which has been described has no chronic form, since, although bronchitis may be chronic, the results of the pleurisy, in opposition to the inflammatory process, are lasting. It is unusual for the acute attack to be continued into a chronic bronchitis; on the other hand, an eventual agglutination of the pleural surfaces, and especially a sealing up of the diaphragmatic groove, are fertile sources of recurring and ultimately of permanent bronchial trouble, in the shape of a localised basic catarrh. Of all local bronchial catarrhs the most common is the apex-catarrh or phthisis, or the recurrent simple apex-catarrh so often determined by the indurated and adherent scar of an old tuberculous lesion. In both cases the same mechanical influence is exerted by the adhesions in hampering the pulmonary movements and in interfering with the systematic play of the expiratory currents.

At the base, and particularly at the lateral base, distinguished in health by its active inspiratory movements, the local catarrh is apt to lead to extensive tissue changes. It is customary to speak of the affection as a "chronic pulmonary catarrh," and of the ultimate anatomical condition as a "chronic interstitial pleuro-pneumonia." We should not lose sight, however, of the essentially bronchitic origin of the mischief. The localisation and the permanence of the catarrh are primarily due to the paralysing influence of the adhesions. The combined irritations exerted within the air-passages by the retained secretion, and without by the recurring respiratory traction, may set up a purely secondary fibrosis; and in some cases the fibrosis is mainly perilobular. Sometimes, however, the affection remains to the end essentially bronchitic with a tendency to rarefaction rather than to condensation of the pulmonary substance. Further consideration will be given to this subject in the article on "Bronchiectasis."

E. PLASTIC BRONCHITIS

This curious and rare disease, referred to by Galen and studied in 1697 by Clarke and Lister, has been repeatedly described since that time. Biermer deals with a series of fifty-eight reported cases; but Peacock had previously given the first collection of cases on record. Lebert treats exhaustively of the same subject. Dr. Samuel West has collected fifty-two cases recorded since Lebert's article, and compiled a full bibliography. Plastic bronchitis, according to Biermer, occurs twice as frequently in the male as in the female sex, but is not confined to any age from early infancy to advanced life; though most commonly observed in the intervening period. It is still a pathological enigma.

The membranous exudations sometimes occurring in the air-passages form a large and varied group. False membranes may originate from the action of strong fumes or irritating fluids. The inhalation of steam (Parker), or of the fumes of ammonia, or of alcohol in the shape of eau-de-Cologne, are well-known instances. Again, the introduction into the air-passages of strong solutions, such as lactic acid, has been followed by plastic exudation (cf. Hoffmann); and Fritzsche describes a case in which he attributed the latter to the internal use of iodide of potassium.

As a result of disease, thin false membranes have been observed in the bronchi not only in instances of diphtheria, phthisis, erysipelas, variola, scarlet fever, measles, typhoid fever, and sewer infection (as in the cases of Picchini, quoted by Magniaux), but also in ordinary bronchitis, or pneumonia (R. Koch), in various pulmonary and cardiac diseases, in articular rheumatism (Degler), and in pemphigus (Mader).

From all these varieties of membrane, as well as from the rarer forms which have been described as primary diphtheritic and primary pneumococcic (Magniaux), the membrane of plastic bronchitis differs in its greater firmness, which allows it to be expectorated in considerable arborescent masses. The casts occasionally brought up after hæmoptysis could alone compare with the latter in size and in consistence, but their origin and their composition are both sufficiently manifest. Thus whilst presenting distant affinities with the minute bronchiolar and sometimes the coarser bronchial plugs of pneumonia, with the tubular casts of diphtheria and of membranous tracheitis, and even with the occasional intratubal mucous inspissations of acute bronchitis seen chiefly in children, the formation of a continuous arborescent mould of a considerable portion of the bronchial tree stands by itself as a well-defined, although hitherto unexplained pathological process.

Whether this feature may be trusted as a sufficient indication of the pathological individuality of the affections is doubtful. Plastic bronchitis may possibly be not always of the same kind; it may be due to a variety of causes, just as there are distinct varieties of pseudo-membranous affections. Again, the fact that most of the latter have been traced to a bacterial origin, suggests that a similar causation may at some future time be made out in plastic bronchitis. In spite of this uncertainty as to the unity and as to the mode of origin of the latter, we note in the cases a general agreement which binds them together into a distinct nosological group characterised anatomically by the recurring exudation, both rapid and extensive, of coagulable material in the bronchial tubes, coupled, it is said, with denudation of epithelium, and nosologically by the mechanical results of the exudation, namely, paroxysmal dyspnœa and the partial or total expulsion of the casts; or, in the more severe cases, suffocation and death.

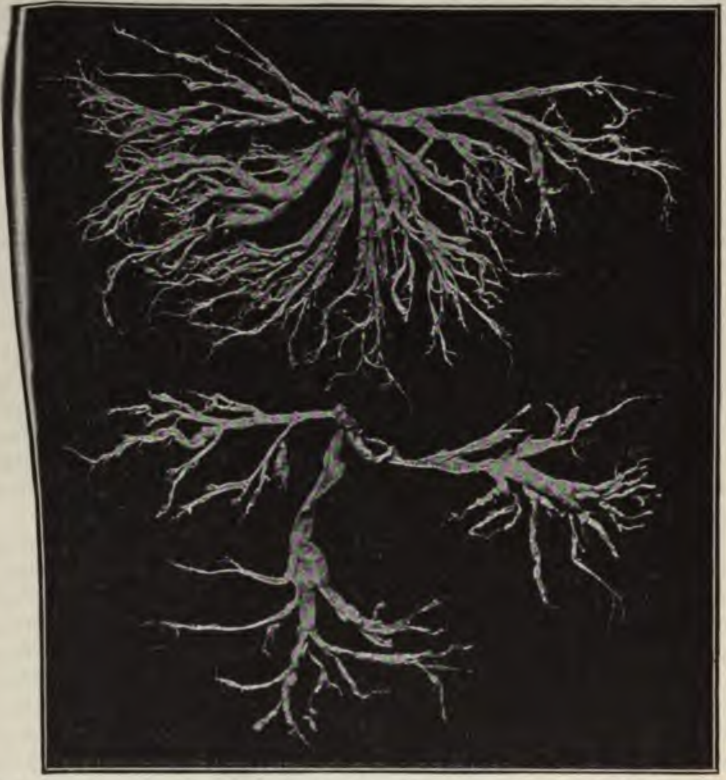

Fig. 1.—Casts expectorated by two patients suffering from plastic bronchitis. For an account of the cases see *Catalogue of St. George's Hospital Museum*, Series vii. 30A, 30B. (Size considerably reduced.)

Clinically the disease is clearly distinct from any of the affections enumerated, occurring rather in connection with some personal idiosyncrasy than under the influence of any recognised predisposing circumstance, diathesis, or disease, and affecting robust subjects as well as those suspected of actual or of threatening tuberculosis. It was observed by Oppolzer recurrently during menstrual periods, with intermittence during pregnancy; whilst Biermer records several cases occurring during pregnancy (Wilson Fox). In its exciting causes—climatic, seasonal, and others—it is closely analogous to common bronchitis, and is in its beginnings almost indistinguishable from the latter, the exudation supervening upon an initial catarrh.

The symptoms are those which would ensue from any extensive obstruction of air-tubes. The cough, which may have a peculiar tone, varies in intensity with the extent and consistence of the recurring thrombi; when they attain considerable size and extension their expulsion is preceded by hacking dry cough and dyspnœa lasting for hours, and in the expiratory type of the spasms resembling that of asthma. The cyanosis is usually moderate. Permanent dyspnœa is present in the proportion of the existing obstruction: during the intervals of freedom from membrane it is not complained of. Slight hæmoptysis, more often following than preceding the expulsion of the casts, is very frequent in nearly one-third of all cases (Biermer); or in one-third of the acute cases (Lebert). Sometimes it is considerable; and this has given rise to an opinion that the casts might consist merely of coagulated blood; but they contain no blood-discs, except in their outermost layers, which are frequently streaked with blood (Wilson Fox, Biermer).

Ordinary mucous sputum is apt to alternate with the casts, or to accompany them throughout when they are expectorated piecemeal; and a mucous expectoration precedes the expulsion of the larger masses, which are commonly ejected balled up in a slimy investment. Five to ten days is the most common period of retention of the casts; but this may range from one or two days to upwards of three weeks. The daily expectoration of casts may be considerable for long periods, or limited to a few fragments for a few days (Wilson Fox).

The constitutional symptoms in average cases are slight; including little or no pyrexia, except in the early stage, but occasionally a recurrent pyrexia with rigors, little emaciation, and, in a few cases only, dropsy, epistaxis, diarrhœa, or albuminuria, which may not exceed the duration of the attack. The spleen is sometimes enlarged.

A convenient division has been made between a small group of cases running an acute course (from one to four weeks or more), and a much larger group of chronic course, extending over years with intermissions and relapses of varying durations. Biermer again subdivides the acute cases into a mild variety, of shorter duration, in which the ordinary symptoms of a slight bronchitis are simply varied by the expectoration of a few casts; and a severe variety, pyrexial and suffocative, in which death may occur (six cases fatal in a series of ten) before any of the casts have been expelled. The chronic form may long simulate ordinary bronchial catarrh; or it may declare itself early. It also resembles bronchitis in its relapsing character.

The physical signs, ill-defined where the plugs are small and few, are well marked in cases of extensive obstruction. Inspiratory retraction of the chest may occur. At any rate the respiratory movement is locally impaired; and pulmonary collapse may give rise to dulness, whilst full or exaggerated resonance is elsewhere obtained. The respiratory murmur is diminished or absent; or it may be replaced by sibilant râles, by moist râles of various sizes, and, on the coagula becoming loosened, by loud whistling (Corrigan), by tubular breathing and coarse râles (Van Meerbeck), by a peculiar valve sound (Barth and Cazeaux), or by various flapping sounds described by German authors as *schmetterend*, *schnarrend*, and *flatter-geräusch* (Hoffmann). On palpation a tactile fremitus may also be felt, which has been attributed to the flapping of the bronchial casts.

Prognosis.—The association of the disease with tuberculous phthisis in a certain proportion of the cases somewhat artificially raises its mortality. Putting aside this latter group, and the unusual instances with severe onset and rapidly fatal tendency, in which grave dyspnœa coinciding with scanty expectoration and with extensive collapse of the lung are the most anxious features, the disease, as generally observed, "neither destroys life nor does grave damage, general or local" (Walshe). The liability to attacks may last for considerable periods. The case recorded by Kisch extended over twenty-five years.

Morbid anatomy.—(i.) The casts may be expectorated in mere fragments or in their unbroken state. When freed from mucus, by suspension in water, undamaged specimens are found to reproduce the structure of the bronchi, from the tubes of the diameter of a goose-quill (rarely of much larger size) down to the finest ramifications, with such perfect accuracy that the site of their formation can be readily identified by comparing them with a cast of the bronchial tree obtained by artificial injection and corrosion. They are, with the exception of the smaller branchings, of firm consistence, and often perceptibly tubular; their bore being commonly plugged with mucus. "Their colour is whitish or pearly gray. They are distinctly stratified, and consist of a structureless or fibrillated basis in which are embedded inflammatory cells, mucous corpuscles, pus cells, pigmented cells, and altered gland cells, and, in their outer layers only, blood cells. They are soluble in alkalies, and also in lime-water" (Wilson Fox).

The expression "plastic bronchitis" does not define the nature of the exudation; and in this there is an advantage, since the casts are invariably mixed products, and may consist largely of mucus, as shown by the action of the solvents just mentioned. Nevertheless they are mainly fibrinous, and owe to fibrin their characteristic consistence. Of this an indirect proof is found in the great rapidity with which fresh casts may be formed after the expectoration of previous ones. Waldenburg, and subsequently P. Lucas-Championnière, had described the occasional presence of fat in the casts. This observation has been confirmed by Model, who finds that the fat occurs as a fine granular deposit, or in droplets between layers of fibrin; it is sometimes to be seen floating in the sputum, which may contain so much of it as to suggest an escape of lymph or of chyle from the bronchial membrane.

Among the formed elements detected by the microscope in the casts should be mentioned bacteria, and occasionally hæmatoidin crystals, Curschmann's spirals, and particularly Charcot Leyden's crystals and eosinophilic cells.[1]

(ii.) The bronchi after death may contain casts in place, or imperfectly solidified curdy collections; or they may be quite clear and present catarrhal mucus only. The membrane may be injected, or pale, as in Biermer's case, in which the epithelial lining persisted under the cast. In Kretschy's instance of an exceedingly rapid reproduction of the casts, the bronchi affected were deprived of their epithelium, and it was evident that the casts were not due to desquamation and transformation of cells, but to a genuine outpouring from the blood-vessels or lymphatics.

Emphysema is almost invariably present. Cases are sometimes cut short by intercurrent acute bronchitis or pneumonia. Traces of pleurisy, recent or antecedent, are sometimes found. Tubercle is present in a small proportion of the fatal cases. Model has recorded its occurrence in 10 cases in a series of 21 cases of the affection. Dilatation of the bronchi has been very rarely found. Mader attaches some etiological importance to the coincidence of pemphigus with plastic bronchitis.

The diagnosis can only be made after the expectoration of some of the coagula. The characters special to the latter, when recognised on examination, should enable us to distinguish the case from cases of intrabronchial hæmorrhage and clotting, of diphtheria, of acute bronchitis, and of asthma.

Treatment of an effectual kind has yet to be discovered. The solubility of the casts in lime-water, originally discovered by Dixon, which strongly suggests the presence within the casts of a large proportion of mucin, led Biermer to recommend the inhalation of atomised lime-water, and a case of its successful employment has been reported by Waldenburg.

The only other rational treatment which has been specially recommended is the use of emetics.

The natural process of catarrh by which the plugs are loosened tells in favour of the emollient action of an atmosphere of vapour. This measure, strongly advocated by Walshe, has the advantage of being harmless; and Dr. Ogle suggested that the steam might be medicated with tar or with other stimulating ingredients. Iodide of potassium, internally, was favourably spoken of in 1854 by Thierfelder and Wunderlich. Creasote, tar, turpentine have also been advocated; and Biermer recommends the free administration of mercury in acute cases.

The ordinary treatment of bronchitis is suitable for the generality of cases; and this applies also to the climatic indication, in spite of the disappointing results which have been reported.

An important precautionary measure in connection with the severe dyspnœa to which such patients are liable, is to provide, in all ascertained or suspected cases of the disease, a readily available, if small, supply of oxygen for immediate use in the event of a sudden difficulty of breathing; whereby time may be afforded for procuring more abundant supplies, and for the adoption of other measures of relief.

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[1] For references on these and on various other points the writer is indebted to Professor Hoffmann's valuable article, "Die fibrinöse bronchitis."

Among the latter I would also suggest in future cases the trial of two other rational methods of treatment. The local treatment of the mucous membrane and the removal of the casts are clearly our first indications; and we are now in possession of a method by which they seem likely to be fulfilled, namely, the cautious intratracheal injection of *oil* or of some *mild solvent*. These forms of treatment have not yet, so far as I am aware, been resorted to in plastic bronchitis; but I believe that they may ultimately be found more successful, in promoting the expulsion of the casts and in obviating their recurrence, than any less direct method hitherto adopted.

Had we a free choice, our preference would be for some safe means of speedily and completely detaching the bronchial cast; and on applying the remedy we should be aiding nature by following her own method. We might even improve upon the latter if the agent employed could exert some healing action upon the damaged mucous surface. Oil may prove to fulfil both requirements. Its non-irritating character, its power of penetration, and its property of rapidly spreading over, and of protecting moist surfaces, even when used in small quantities only, are important recommendations. The intratracheal method suffers from the lack of any means of regulating the course taken by the injection and of ascertaining whether the latter comes more into contact with the healthy mucous membrane or with the casts. In the case of oil this is happily an unimportant objection: we can trust it to find its way wherever any space offers. At the same time there remains, even in connection with its sparing use, the important reservation that the air way is already greatly obstructed, and that any form of injection might aggravate the dyspnœa.

Some support is given to this suggestion by the happy result obtained in a case of diphtheria, where obstruction of the trachea with membrane was set up after a previous tracheotomy. Creasoted oil (1 in 20) dropped at intervals through the tracheotomy tube excited the desired amount of cough, and enabled the membrane to be expectorated with remarkable facility, so that the case ended in recovery.[1]

The notion of breaking up the membrane agrees less closely with rational principles and with the lines of the spontaneous process of cure. Whichever be the solvents selected for injection, their concentration has to be slight; their action will therefore be slow, and their bulk must be relatively large. Moreover, their influence upon the diseased mucous membrane itself is an anxious question. Above all, we cannot forget that our object is the removal of the plug rather than its destruction. Integrity of the bronchial casts is an important help towards its complete expectoration: its solution piecemeal might be a doubtful gain, if the smaller branches of the cast were to be left behind.

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[1] Favourable results, in obstruction of the trachea by diphtheritic membrane, from the introduction of creasoted oil through the tracheotomy tube, by William Ewart, M.D., F.R.C.P., and W. A. Hubert, L.R.C.P., M.R.C.S. *Brit. Med. Journal*, Nov. 27, 1897.

Among the solvents at our disposal, lime-water would probably be the one least open to objection, putting aside the serious risk connected with the bulkiness of the injection. Lactic acid and the digestive ferments, which Dr. Rolleston has suggested to me as alternatives, are perhaps not equally suitable. Lactic acid has been credited with setting up pseudomembranous bronchitis when accidentally dropped into the trachea, and its employment even in dilute solutions might be open to question. The digestive ferments have been tried in diphtheria of the fauces with very unequal results. Trypsin is free from the chemical objection which may be urged against pepsin, and to a slighter extent even against the vegetable ferments papayotin and papain, which act best, though not exclusively, as does pepsin, in acid solutions. The results obtained with papayotin in diphtheria were not encouraging. The favourable opinion entertained by Rossbach has not been shared by other observers, the solution of the ferment having been either too dilute to be effective, or, when of a strength sufficient to destroy the false membrane, not free from damaging effects upon the mucous surface. Papain itself, the more powerful product yielded by the fruit of *Carica Papaya* (papayotin being derived from the milky sap), has been recommended; but the success of its employment, even with the advantage of the relative accessibility of the surface to be treated, has not been such as to bring it into general use. The effects of the intratracheal injection would need to be studied experimentally before it could be confidently recommended, since any advantage might be outweighed by the slightest irritation set up in the mucous membrane.

In conclusion, the suggestion of a local treatment of the affection, whilst opening up a promising therapeutic prospect, may prove in the event impracticable. In any case the attempt to carry it out should be made with the utmost caution. The local treatment by bactericidal agents and the hypodermic treatment by antitoxins are possibilities contingent upon the results of future pathological discovery.

F. PUTRID BRONCHITIS

In the course of an inveterate purulent bronchorrhœa the expectoration occasionally becomes putrid; and to this condition in its worst form the name putrid bronchitis has been applied. Putrid expectoration occurs in bronchiectasis, and is commonly associated with destructive pulmonary lesions. Instances of the uncomplicated kind are comparatively rare, the majority of the cases occurring as a late complication of long-established bronchial dilatation.

If foulness of the expectoration in itself constituted a putrid bronchitis, we might group under that name, together with many cases of bronchiectasis, all cases of pulmonary gangrene, of gangrenous tuberculous phthisis, and of putrid empyema discharging through the lung.

All these, however, are removed into other categories by reason of the prevailing importance of their extra-bronchial lesions. It is to the remaining cases, in which the bronchial trouble either stands alone or largely predominates, that the name strictly applies. Although even here the affection is seldom, if ever, absolutely primary, the pre-existing catarrh, emphysema, pleuro-pneumonia, interstitial pneumonia, or fibroid degeneration, whilst they account for a delayed expectoration of the bronchial contents, do not in themselves explain their putrid decomposition. The cause of the latter is intra-bronchial; and two views have been taken of its etiology. According to some bacteriologists putridity is mainly due to the influence of micro-organisms, and the bronchitis is secondary to the microbian invasion—a view to which we shall presently refer. Other pathologists have regarded the bronchitis as the primary event, and have sought to trace the sceptic process to definite structural changes in the bronchial mucous membrane.

The association of gangrene with bronchiectasis had been dwelt upon by Laennec. It was more definitely described in 1841 by Briguet as affecting the terminations of the dilated tubes. Marfan has recently endeavoured to apply the same explanation to putrid bronchitis. He assumes the existence of a primary *gangrene of the bronchi* which, he contends, attacks the middle-sized and smaller tubes independently of any bronchiectasis, or in association with but small terminal dilatations. Additional evidence will be needed before this view can be regarded as proved. Meanwhile it is significant that lesions of this kind have not been noticed by other observers; and that in one case in which they were specially looked for after death they were reported, by Sée, to have been absent.

The view more generally accepted is that an ordinary bronchitis may degenerate into the putrid form, which may or may not be a merely passing phase, but cannot persist for long periods without progressive damage to the bronchial structures and serious risk to life.

That putridity may be set up within the air-tubes by the inhalation of septic matter is a possibility suggested by cases such as that of Tiedemann, in which this was brought about by a leakage into a pulmonary cavity from a traction diverticulum. The attempt to attach the blame to any individual variety of micro-organism is rendered difficult not only by the number of microbes gaining access to the bronchi, but also by the necessity of explaining this occasional failure of the protective mechanisms which normally succeed in repressing them even in cases, for instance those of phthisis, apparently most liable to infection.

The bacteriology of the sputum has already grown to considerable proportions. Among the numerous micro-organisms discovered in putrid expectoration, several of which have been cultivated, J. Lumniczer has succeeded in isolating a bacillus which perhaps may be the same as that isolated by Bernabei, giving, after a few days, the same odour as the sputum. Bernabei is inclined to regard putrid bronchitis as directly due to the growth of the specific bacillus which he has described. Hitzig has likewise described two bacilli not unlike the bacilli coli communis, also yielding a fetid odour.

The inhalation of oidium albicans was regarded as the cause of the affection in an isolated case reported by Rosenstein; and Canali has reported a case in which actinomycosis was either a cause or a complication.

The sputum sometimes presents a brownish discoloration; it is intensely fetid, either of gangrenous or of foul, sweetish odour. It separates into three layers—an upper muco-purulent and frothy layer, a middle translucent opalescent layer, and a lowermost dirty, yellowish, granular layer containing the solid constituents which have been deposited. As far back as 1850 Dittrich had described the plugging of some of the bronchial tubes by small friable masses, varying in size from that of a millet seed to that of a bean, made up of cellular débris, pus cells, granules, oil globules, hæmatoidin crystals, and various micro-organisms, including the monas and cercomonas described by Kannenberg and by Streng, and leptothrix pulmonalis. These "Dittrich's plugs" make their appearance in the expectoration and, together with the intensely fetid odour, settle the diagnosis. Fatty crystals (palmitic and stearic), volatile fatty acids (valerianic and butyric), leucin and tyrosin, methylamin, ammonia, and sulphuretted hydrogen are also found. Leptothrix pulmonalis occasions a purple, violet, or blue discoloration of the sputum when treated by iodine, a reaction observed by Virchow and by Gamgee. Jaffé's observations of the presence of minute quantities of leucin and of tyrosin are of interest in connection with the ferment obtained from the sputum by Filehne and by Stolnikow, which they regard as analogous to pancreatic ferment. The same observers confirm the observation that Dittrich's plugs contain a substance striking blue with iodine.

Morbid anatomy.—Pathological changes special to the affection are comparatively few. The post-mortem appearances are those of an intense bronchitis and peribronchitis with pneumonic infiltration of the surrounding tissue. Pneumonic consolidation may be found extending over more or less extensive patches; but the greater part of the lung is in a state of congestive and puriform œdema, and the bronchial glands are swollen and soft. Some of the bronchi may show ulceration, or the mucous membrane is softened in places and deprived of its epithelium; or it may become involved with the adjoining pulmonary tissue into genuine gangrene. Cases of this kind have doubtless supplied Marfan with the basis for his separate description of a gangrene of the bronchi. The collateral changes are varied according to the morbid antecedents of each case.

The symptoms accurately given by Dittrich consist in a sudden onset of feverishness soon assuming a typhoid character, intense depression, collapse, coma, and death. The attack is accompanied or preceded by an equally sudden change in the sputum, from the habitual muco-purulent type of chronic bronchial catarrh to the putrid variety. At the approach of death expectoration diminishes and finally ceases.

The long paroxysmal coughs peculiar to advanced excavating disease, and the gushes of sputum of bronchiectasis are not witnessed; but the frequency of the cough and of the expectoration is often severe, particularly in the pleuro-pneumonic and fibroid cases, in which the thoracic excursions are much restricted. Fever of a remittent type is usually present throughout the putrid stage; and it may be regarded as a measure of the septic absorption.

The **diagnosis** is based upon the clinical history and upon the negative results of a physical examination for the lesions of bronchiectasis, of phthisis, and of pulmonary gangrene.

The **prognosis** varies according as the putrid condition of the bronchial contents is grafted upon a simple chronic catarrh, or is combined with deep-seated tissue irritation and overgrowth. In the first group of cases recovery may take place after a few weeks, but relapses will be apt to occur. In the second group the fatal tendency may be hastened by catarrhal pneumonia, acute bronchitis, pulmonary gangrene, pleurisy, metastatic abscesses (including cerebral abscesses), or endocarditis.

TREATMENT OF BRONCHITIS.—Some account has been given of the treatment of plastic bronchitis: that of putrid bronchitis will be dealt with under the heading of Fetid Bronchiectasis. The other varieties of bronchitis will now be considered in turn from the point of view of the abortive, the curative, and the palliative treatment; and of prophylaxis.

I. Trachea-bronchitis.—(*a*) *The abortive treatment* of simple chest cold, at its preliminary stage of coryza, has probably been more often attempted than in any other ailment, and with a greater variety of methods, most of which are based on diaphoresis and diuresis. The suppression of the coryza has, however, been sometimes attempted by a direct local action on the mucous membrane of the upper air-passages, or by way of the nervous and vaso-motor system. The direct application of powders or snuffs variously compounded of quinine, camphor, subnitrate of bismuth, morphine and astringents, and the inhalation of stimulant camphorated vapours, in which ammonia, carbolic acid, iodine, and essential oils are prominent ingredients, have often been prescribed; and remedies of this kind have at times been advertised as specifics. Of internal medication two methods have been used—the tonic and the sedative; on the one hand, liberal doses of quinine or, as strongly advocated by my friend Dr. Isambard Owen, of the tincture of perchloride of iron; on the other, large doses of potassium bromide or some of the antipyretic remedies recently brought into use, especially phenacetin.

The diaphoretic methods do not need any detailed description: they include the traditional help of a Dover's powder, of hot grog, of blankets, of the hot air or Turkish bath, or of the vapor bath, a medicated modification of which has enjoyed some reputation in country districts. In practising this homely and doubtless efficacious method the patient stoops over a vessel of boiling water in which are infused a quantity of selected herbs, while the body is entirely covered with a sheet or blankets; after a few minutes' inhalation of the aromatic vapour profuse perspiration is induced, and a cure may result. The late Sir Andrew Clark's favourite diaphoretic treatment was the hourly administration of ammonium citrate assisted by warm drinks and warm wraps.

(*b*) *The curative treatment.*—If in spite of all efforts trachea-bronchitis should advance, its rapid relief can only be secured by rest in bed, fluid diet, warm drinks, and assiduous treatment beginning with a quickly acting purge and combining diuretic and sedative action with the all-important diaphoresis. A hot foot-bath with the addition of mustard, a mustard poultice to the front of the chest, and the inhalation of steam medicated with terebene, eucalyptol, or the compound tincture of benzoin, are valuable adjuncts to the treatment. As soon as decided improvement becomes manifest, iron and quinine should be substituted for the saline remedies, and the ordinary diet resumed.

II. Simple acute bronchitis of the middle-sized tubes. — This affection, though not usually dangerous, calls for judicious and active treatment. The preliminary measures are directed to the relief of congestion of the liver and of the alimentary canal by two to four grains of calomel followed in an hour or two by a black draught. Meanwhile arrangements are to be made for the regulation of the temperature of the room, at a mean of about 65° F.; for the occasional supply of steam, and for its medication with eucalyptus, thymol, or wool-fir oil. The delay before purgation may afford time for a foot-bath or for the application of mustard leaves to the calves, to the upper sternum, and to the shoulders, — or of dry cups over the back. Blisters are unnecessary, and may be inconvenient during the subsequent perspiration. The patient should be kept in bed and allowed to assume the position of greatest comfort, probably one of slight elevation of the head and shoulders.

The more quickly *diaphoresis* can be obtained the greater will be the hope of checking the spread of the bronchitis. The wet-pack is sometimes used; but more generally the vapour-bath will be preferred, some form of which may easily be improvised. Internally the administration of acetate of ammonium, spirits of nitrous ether and of chloroform, with syrup of squills or of red poppies, and infusion of senega, will be found useful together with other means of keeping up the perspiration. In more active inflammation nothing will relieve the tightness at the chest and the hardness of the cough, whilst reducing arterial tension and keeping the skin moist, better than antimony. Relatively small doses (not exceeding 8 ♏) of antimonial wine, combined with small doses of Dover's powder, or of bimeconate of morphine, the tendency of which is likewise to relax arterial and bronchial spasm and to reduce active congestion, afford much relief. A fluid diet of beef tea, milk, gruel, and tea of moderate strength, belongs to this stage; and, when administered warm, adds much to the action of the treatment.

Expectorants. — Antimony, used as indicated above, undoubtedly loosens the phlegm and promotes its expectoration; but a different combination is called for as soon as the initial discomfort has been allayed, and the skin, kidneys, and liver have been thoroughly brought into action. It is now time for the direct expectorants—squills, ipecacuanha, carbonate of ammonium, and especially potassium iodide, which, in cases presenting much spasm of the air-tubes, may be successfully combined with the ethereal tincture of lobelia and spirits of chloroform.

Belladonna, one of the early remedies for bronchitis, has not permanently held the position repeatedly claimed for it. This suggests that it may not be equally suitable to all cases, and that in some instances the adjustment of the dose may be a matter of unusual importance, as might well happen with a drug possessing several powerful physiological actions. Each of the latter has in turn been credited with the remarkable results reported by observers. As the element of bronchial spasm in varying degrees enters into all cases of bronchitis, belladonna would be more useful where this factor more largely prevails; as in the bronchitis of asthma and sometimes of emphysema. In cases of this kind the remedy has, in my experience, occasionally afforded more relief when worn as a plaster over the chest than in the form of internal medication. Recently Dr. Sydney Ringer has recalled our attention to its efficacy in bronchitis in relieving the incessant cough and checking the flow of mucus, which, whether viscid and scanty or profuse and watery, is regarded by him rather as an increase of secretion than as an inflammatory product. He prescribes 10 ♏ doses thrice daily or oftener. On the strength of the same property of checking the secretion he suggests its employment in ether bronchitis, and in those cases in which aspiration of the chest is followed by a profuse and sometimes suffocating amount of expectoration. The value of belladonna is also advocated by Dr. Murrell, who points out that the same advantages may be obtained by a solution of homatropine. Lastly, Mr. Davies of Sherborne has dwelt upon its "magic" effects as an inhalation, not only in asthma but in acute bronchitis. He recommends the use of 1 grain of the extract in ½ oz. of water with Siegel's inhaler, which has the additional advantage of moistening the atmosphere.

Inhalations.—To allay the irritable cough conium or chloroform may be added, in the steam inhaler, to representatives of the turpentine group, such as thymol or eucalyptol; but, for the relief of spasm of the smaller tubes, the dry inhaler[1] is usually found more effectual. It consists essentially of a Woolffe's bottle, provided with a long inhaling tube and mouthpiece, and packed with tow or loose cotton-wool steeped in volatile principles which impregnate the air inhaled through the bottle. The chief sedative agent to be used in all the mixtures for inhalation is undoubtedly spirits of chloroform; the other constituents may be freely varied according to indications and to suit the patient's taste.

Emetics and bleeding, formerly much in vogue and regarded as almost indispensable, are still not infrequently resorted to in some European countries; but they have long been neglected in England. Against this neglect Dr. C. J. Hare has raised an energetic protest. He has specially insisted on the great value of emesis in acute bronchitis. In addition to its general and hepatic action, it not only removes the existing accumulation, but, by its mechanical effect, squeezes out of the mucous membrane a large quantity of the effete cellular and mucous material, thus warding off the danger of an implication of the smaller tubes. Its early employment before the onset of this complication would be free from the risk of overtaxing the heart at a time when recovery in great measure depends upon the cardiac energy being fully sustained.

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[1] For a fuller description of this apparatus by the writer, *vide Clinical Journal*, 21st Dec. 1892.

Bleeding was prescribed early in the attack in bygone days. At the present time it is not systematically used as a prophylactic, but is reserved for any more urgent symptoms which might supervene. The treatment of the catarrhal muco-purulent stage of this milder form of bronchitis is practically the same as that of chronic bronchial catarrh, and to that section the reader is referred.

III. Acute suffocative bronchitis of the adult.—In all cases of severe bronchitis, or in any case of mild bronchitis threatening to become severe, the first and all-important indication is to provide a ready supply of *oxygen*. In a dilute form oxygen cannot fail to be of use even before the onset of urgent dyspnœa; and it cannot do harm. When dyspnœa has set in, the amount of relief its undiluted administration will afford is limited only by the difficulties of respiration. The objection that the air-distended chest and the choked bronchioles often refuse to inspire has led some authors to regard the treatment by oxygen as useless; but this should not discourage our efforts, for we must bear in mind how relatively small is the bulk of oxygen which corresponds to the ordinary intake of air: during the stage of greatest severity the inhalation of oxygen in some form or other should be maintained continuously. This method tends to fulfil two needs, the pulmonary and cardiac. The excellent results of the local treatment of cutaneous ulcers by an atmosphere of oxygen as originally prescribed and practised by Dr. George Stoker would lead us to expect a like beneficial action upon the mucous membrane. But the second is perhaps the more important function: final success in a protracted and severe struggle for breath is directly dependent upon the vigour of the heart and upon the endurance of the respiratory muscles. Any improvement in their effectual working will tend to increase the subsequent intake of oxygen, and with it the cardiac and thoracic energy.

Another therapeutic agent of importance is moisture supplied as *steam*. Its application, however, needs care. An excess of steam, or still worse, of the heat directly due to it and to the lamp or fire used to raise it, is injurious. Steam and oxygen work well together; the dryness of the oxygen is tempered by the steam, and the depressing effect of the vapour is relieved by the stimulation of the gas.

In the *medicinal* treatment three objects must be kept steadily in view from the first, all being urgent:—(i.) to keep up the patient's strength; (ii.) to relieve the bronchial spasm as much as possible; (iii.) to mature, that is, to loosen the catarrh. A preliminary dose of calomel, followed by a saline, will do good in every way; but this is the full extent to which any depressing treatment or methods involving exertion on the part of the patient can be countenanced. The question of an emetic should, however, be considered, and will need much judgment. This remedy is one exclusively for early employment. A large jacket poultice, made as light as it is possible in front, is of distinct advantage.

Alcoholic stimulation.—Whenever, as in this dangerous malady, the patient's safety lies in the correctness of the estimate we can form of his vital powers as a guide to treatment, the worst evil would be a delusive aspect of strength: the early and over-zealous administration of alcohol entails this risk. Although in certain cases a need for alcoholic stimulation may seem to exist from the first, let us bear in mind that alcohol is not curative in suffocative bronchitis; it should not be our first resort, but be brought in rather as a powerful reserve to carry a desperate position or to ensure its being firmly held. It is impossible, of course, to lay down any general rule as to the time for the employment of alcohol. The physician's estimate of the actual and prospective store of cardiac energy in the individual case is the best guide.

Cardiac stimulants.—Meanwhile, however, cardiac stimulants are to be administered without any delay. A mixture containing carbonate of ammonium in sufficient amount, liquid extract of cinchona, iodide of potassium (3 grains), and antimonial wine (3 ♏), with syrup of squills and senega, may be administered every two or three hours at first. A few doses of the following mixture may also be at hand for separate administration: 15 to 20 drops of tincture of digitalis, 5 ♏ of liq. strychninæ, 20 ♏ of sulphuric ether or of aromatic spirits of ammonia, with compound tincture of lavender or some other excipient. A dose or two should be prescribed against the risks of the night, and may be given at suitable intervals or under special indications in the day.

The frequency of the administration of the expectorant is modified according to the progress of the case; and an occasional intermission of it, with some cooling acid draught as a substitute, may be welcome to the patient. Perceptible amendment should be noticeable within the first twenty-four hours. In the worst cases it will not be a discouraging result if the patient has done no more than maintain his strength.

Mechanically aided expiration.—As previously stated, the existence of emphysema is a specially dangerous factor, and may call for something more than medicinal treatment. I have found decidedly good results from mechanical assistance to expiration; this may be carried out by the attendant, who places his hands, well spread out, over the axillary bases of the patient's lungs, and exerts very carefully timed pressures, judiciously adapted to the phase of spontaneous expiration. The special appropriateness of this method rests on the fact that in emphysematous cases an important part of the dyspnœa and impeded expiration is dependent upon the inherent weakness of the elastic fibre of the air-sacs, over and above the mechanical obstacle produced within the small tubes by the viscid secretion. The larger the share of the first of these two factors in the individual case, the greater will be the relief obtained. The method may be tried in all cases, but requires to be used with considerable discretion, and with due regard to the patient's feelings, and the effect produced upon the depth and frequency of breathing.

A case cannot remain stationary at this stage; if it do not improve it is rapidly deteriorating, and at any moment, owing to progressive congestion of the right heart, exhaustion may set in. Our duty is to apply the only adequate remedy, venesection, without waiting for the manifestations of extreme cyanosis, cold sweats, jactitation, and fluttering pulse. Direct puncture of the right auricle is for obvious reasons impracticable; but the next best means to a sudden and ample depletion of the cavity is to open the external jugular vein, from which 8 to 10 oz. should be boldly abstracted. The benefit obtained is immediate and considerable; the duration of it will depend upon the degree of remaining cardiac energy.

At this moment alcoholic stimulation, if not previously pushed with improvidence, should prove a boon. This is also the time to bring every cardiac tonic to bear, and to inject, if necessary, under the skin $\frac{1}{30}$th to $\frac{1}{20}$th grain of strychnine. Oxygen, if it had been discontinued, should again be inhaled. Any resulting rally in the cardiac and general energy will afford a fresh opportunity for clearing the chest of loose mucus. After a series of mechanically aided expirations the patient should be encouraged to cough up the accumulations; and, by repeating this process, a good deal may be got rid of.

Very shortly after the bleeding—as soon as the respiration has been attended to—the treatment of the right heart should be resumed. It is all-important to save it from a return of its previous engorgement. A liberal supply of india-rubber cups (six to eight) should be applied to the chest simultaneously, utilising any position accessible without undue disturbance to the patient; and each of them should be reapplied in rotation, so that the depleting action may be kept up for a relatively considerable period. At the same time mustard leaves may be used to the calves.

Good results may be obtained from this alternation of the cardiac and of the respiratory treatment, and from the continued administration of digitalis, bark, and ammonia. If all these fail, no other measures, such as electricity in its various forms, will succeed.

The treatment to be followed in favourable cases, after recovery from the asphyxial stage, is analogous to that which has been described under a previous heading.

IV. Capillary bronchitis.—In infants and small children the same dangers have to be reckoned with, but they are complicated with that of pulmonary collapse, which is practically beyond our power of control, and with the yet more uncontrollable pneumonic changes. Fortunately the onset is often less rapid than in the acute suffocative bronchitis of the adult, and affords a somewhat wider opportunity for treatment.

We are acquainted with three measures of primary importance:

poulticing, emetics, and the combined inhalation of steam and of oxygen. Poultices frequently renewed are specially useful in the small chests of children, but it is essential that they should be light. The old practice of the early induction of vomiting is probably the most effectual means of saving life, and is not in itself a source of danger, the act being relatively easy in small children. If the case be seen before the onset of marked respiratory distress the strength will be quite equal to this treatment; and any sign of respiratory retraction of the thoracic base should call for its immediate employment. For threatening pulmonary collapse vomiting is probably the best, if not the only cure. It tends to fulfil two essential needs, namely, the dislodgment of the mucus from the bronchioles, and the inflation of the lobules by the deep inspirations connected with vomiting. This is indeed the safest way of carrying out the method briefly described in the treatment of the adult; namely, that of affording some mechanical assistance to the thoracic and pulmonary movements. Tartar emetic is generally considered to be unnecessarily depressing. A dose of sulphate of zinc, followed by relays of ipecacuanha wine and of lukewarm drinks, is a prompt and effectual agent. Dr. Rolleston has found good results from the hypodermic injection of apomorphine $\frac{1}{30}$ gr. with liq. strychninæ ♏ $\frac{1}{2}$ to prevent collapse.

Steam is readily supplied in sufficient quantity with the help of the steam-tent. The latter should never form a complete investment, but be limited only to the head of the bed, or to three of its sides. It is dangerous to render the atmosphere oppressive. The inhalation of oxygen needs special management in children. No attempt should ever be made to place the tube into the mouth; it is quite enough to direct the stream of gas towards the nostrils. The first tendency to resist the apparent interference is easily got over, and even infants take kindly to the gas when they have experienced the relief it gives. The administration need not exceed more than a few minutes at a time.

Medicinally the lines to be followed are, with some minor differences, almost identical with those indicated for the adult. Belladonna is a remedy not to be lost sight of in the capillary bronchitis of infants and of young children. Dr. March, who is loud in its praise, administers it in minim doses every four hours for infants of six months old, but reduces the dose on the slightest indication of improvement. He ascribes its value to its stimulant action on the respiratory centres; and this is to be set against the objection sometimes made that it checks the action of the skin and the bronchial secretion, both of which we have been taught to promote.

In the capillary bronchitis of old persons neither emetics nor bleeding are admissible under ordinary circumstances. In them treatment must consist in careful feeding and stimulation, the saving of energy, the promotion of expectoration, and constant and judicious nursing. Oxygen is indispensable; and the regulation of the temperature and of the moisture of the atmosphere is also a point of much nicety.

Theoretically, mechanically aided expiration would seem to be specially indicated; but the rigidity of the senile cartilages, although not always so great as might be expected, is an apparent objection to the method. Moreover, the other conditions are not quite simple, and aged patients are often intolerant of any mechanical interference with the thorax.

Among internal medicines the stimulant and balsamic expectorants are specially appropriate, and, up to a certain point, successful. Quinine or caffeine may have to be associated with carbonate of ammonia, although they are not in themselves remedies for the cough. Digitalis and strychnine must also be thought of, and called to aid if necessary. Strong counter-irritation cannot be recommended without reservation, and blisters are not advisable. A milder form of stimulation of the skin may, however, be obtained from the application to the front of the chest of flannel sprinkled with a drachm or two of terebene, which also serves the purpose of an insensible inhalation.

As previously explained, capillary bronchitis at an advanced age is a most fatal affection, and the chief aim and result of treatment may be but a short prolongation of life.

V. Acute gouty bronchitis.—The special form of acute bronchitis occurring in gouty subjects, sometimes as a precursor, at other times as a phenomenon of recession of the arthritic trouble, is apt to be alarming in its onset, and sometimes fatal. The special features are the degree of the pulmonary congestion and the irregularity of the heart. The sudden subsidence of these grave symptoms on the reappearance of the arthritis has suggested the old treatment of applying mechanical irritation to the great toe or other joints with a view to calling back the local inflammation. If this attempt should succeed, pulmonary relief will frequently follow; but the remedy is an uncertain one. Moreover, the bronchitis does not always stand in this relation to the articular paroxysms; it may be independent of them; and it should be borne in mind that its gravity is sometimes the expression of a complicating renal difficulty. The indications in the more urgent stage are stimulation and derivation. Among derivatives the most convenient are mustard foot-baths and dry cups freely applied; whilst a rapidly acting purge, such as calomel and senna, should be followed up by mild doses of colchicum and of an alkali, if no special contra-indication should exist.

VI. Symptomatic bronchitis.—The treatment of the bronchitis associated with the infectious fevers, sometimes, as originally observed by Laennec, throughout their course, does not often call for separate attention. The management of the bronchitis of asthma and of hay-fever, of mechanically induced bronchitis, of the bronchitis of phthisis, and of that incidental to other parasitic diseases, will be considered in other sections of this work.

VII. Chronic bronchitis.—The varieties of chronic bronchitis call for some detail in their several treatment; but for all of them our therapeutic agents may be arranged under four main indications: (i.) the atmospheric treatment, including the climatic; (ii.) the topical, including counter-irritation; (iii.) the medicinal, and (iv.) the constitutional, including the balnear treatment.

(i.) The value of *climatic treatment* is demonstrated by the rarity of chronic bronchitis among inhabitants of more temperate zones, and by the improvement of invalids from the north whilst under the warmer influences. For the larger number distant journeys are impracticable; artificial atmospheric conditions must therefore be devised. The essentials in an artificial atmosphere are purity of the air-supply, freedom from suspended particles, and due regulation of temperature and moisture. A constant renewal of air without oscillations in the temperature, and a proper supply of moisture—the dryness of artificially-heated air being specially noxious in chronic bronchitis—are problems claiming earnest attention in practical hygiene. Evenness of temperature and of moisture, if they can be secured, will enable the chronic bronchitic to spend indoors the periods of more wintry weather, whilst occasionally enjoying exercise in the open during warmer spells. But this after all is merely protective treatment, rather devised for safety than for cure.

(ii.) *Topical treatment.*—Atmospheric therapeutics aim at something more than mere prophylaxis, and are needed in the more active stages. Strictly, the term should be limited to the volatile agents, which can be used to impregnate the air at the normal temperature. Members of the turpentine group—terebene, pinol, cresol, eucalyptol, creasote, tar, carbolic acid, iodine, and the like—are all in some slight degree volatile; though not to the extent observed in the case of chloroform, alcohol, and ether. Chloride of ammonium vapour, supplied by means of a special inhaler, may be combined with some of the vapours enumerated. All these substances may be inhaled in greater concentration when combined with steam, and this method has the most beneficial effect. The practical means of volatilising carbolic acid and other agents at varying temperatures have received much attention from Dr. Robert Lee.

Reference has already been made to the dry inhaler by means of which the more volatile, as well as a slight proportion of the less volatile, substances can be directly inhaled with the inspiratory current. Lastly, the fine atomising or nebulising sprays, for which some excellent apparatuses have recently been introduced, enable us to add to the list of the atmospheric agents almost any of the non-volatile substances, provided they be soluble. Common salt, bicarbonate of sodium, chloride of ammonium, alum, tannin, and various astringents may be thus used as required. A proportion of the spray probably passes the glottis, though doubtless the greater part is condensed on the pharyngeal walls. To this minimum introduced into the lung we cannot fail to attribute a share in the marked benefit obtained; and we recognise in it a first step towards the more vigorous topical treatment by intralaryngeal injections, from which excellent results may be expected in a large number of cases. The laryngeal insufflation of fine powders is less commonly used, and, owing to the ciliary function, it is doubtful whether their action would extend much below the trachea itself.

Counter-irritation is of undoubted value in most forms of chronic bronchial catarrh, for the treatment of the exacerbations. Its usual modes of application are the irritating liniments and applications, such as croton oil, blistering, and the actual cautery. The latter is extensively used in France under the name of "*pointes-de-feu*," for the relief of cough, of local pain, and of profuse expectoration. For the same objects blistering is invariably useful. In *putrid bronchitis* blisters may prove of decided service in checking both the fœtor and the amount of the expectoration; and in those cases where, owing to fibrosis of the lung, Chaplin's treatment by creasote inhalation is not successful, this mode of relief should be tried.

(iii.) *Internal treatment* has regard not only to the immediate relief of the bronchial trouble, but also to constitutional requirements. The list of those drugs which are beneficial to the membrane need not be given in full; their active constituents are usually such as can be exhaled into the lung, so as to take effect on the bronchial membrane. All the derivatives of tar, and tar itself, the turpentines, and the balsams are valuable in the treatment of chronic bronchitis. The more direct expectorants are also sometimes needed, especially when tonics, which are otherwise to be preferred, act as a source of irritation. The preparations of conium, squills, ipecacuanha, senega, in combination with mild salines, will prove of value in these irritable forms; and if there should be much spasm, morphine, belladonna, hydrocyanic acid, lobelia, and like agents may be required. Of the internal remedies taking special effect on the secreting function of the membrane four groups may be especially mentioned: (*a*) Certain balsams, such as balsam of Peru, of tolu, and the compound tincture of benzoin; among the oleo-resins copaiba, and among the tar derivatives creasote and guaicol (to be taken in capsules). These remedies stimulate the membrane and tend to diminish the catarrh. (*b*) Iodine in all its combinations, and particularly as iodide of potassium, has the opposite tendency, and is especially useful when the mucous membrane is dry and the expectoration scanty and difficult, as in the so-called dry catarrhs. (*c*) Sulphur and the sulphides have long enjoyed a reputation for the relief of suppurative conditions, and their checking influence on the profuse muco-purulent discharge of bronchorrhœa and the worst forms of catarrhs is striking. When this can be combined with the tonic effect of a bracing air and with thermal treatment, results may be obtained such as have established the reputation of Harrogate in this country; and of Eaux Bonnes, Cauterets, Luchon, Aix-les-Bains, and other stations abroad.

At all thermal stations patients are subjected to a limited course of treatment by baths, mineral-water drinking, and exercise in the open air. When sulphur is administered to a patient treated at home the same attention should be given to a limitation of the period of administration, lest irritability of the mucous membrane or irritability of the skin should be induced. Lastly, (*d*) cod-liver oil, when tolerated, is an invaluable remedy.

(iv.) *Constitutional treatment.*—As a rule, a slightly purgative plan is of great value; indeed this is one of the favourable aspects of the treatment by sulphur. Various mineral waters may be used, and, with the same object, patients are sent to various medicinal springs.

The cardiac indication is usually obvious. The right heart needs not only to be cured of its dilatation, but if possible toned up. Strychnine, digitalis, strophanthus are thus direct agents in relieving chronic bronchitis by reducing the pulmonary congestion. We should not forget that an excellent way to strengthen the right heart is to strengthen the left. In chronic bronchitis shortness of breath leads to muscular inertia and atrophy; for this there is a remedy in oxygen inhalations, or in their equivalent, systematic purposive hyperpnœa. Patients would gain much by training themselves to breathe to the utmost mechanical advantage, and by cultivating general muscular exercise, at first purely passive, but ultimately active. A general recovery of neuro-muscular energy, other circumstances being favourable, will act most beneficially on the chest through the great improvement in cardiac strength. For artificial methods of lung gymnastics the reader is referred to the chapter on Aerotherapeutics in the first volume.

Lastly, hæmatinic remedies are wanted in a large number of cases; this is a special indication in the groups of protracted muco-purulent and of all severe purulent catarrhs; and these are also the cases which most benefit under cod-liver oil. The administration of iron is not to be limited to those patients whose anæmia and wasting are obvious; iron and quassia, or some other bitter, and particularly cinchona, are not only well tolerated, but of direct value as stimulants to the relaxed and congested bronchial membrane in cases where venous embarrassment gives rise to a deceptive appearance of plethora.

In all cases of inveterate catarrh, but particularly in those which from their severity deserve the name of bronchorrhœa, a warm and equable climate during the winter is indispensable. Various sheltered stations have been recommended in this country, such as the Undercliff, Torquay, Falmouth, Ilfracombe, Minehead, the Scilly Isles, and others. Some patients will derive great benefit from a winter's residence in the bracing atmosphere of Thanet. Nevertheless, whenever this is possible, the Mediterranean seaside resorts are to be preferred; and among them the more sheltered, such as Mentone, San Remo, Alassio, Rapallo, the Riviera di Levante, Capri, Malaga, Corfu, Egypt, and suitable resorts on the North African coast. This large subject is fully treated in the article on "Climate in the Treatment of Disease" in the first volume.

Unless the membrane be protected from irritation for prolonged periods no lasting improvement in the condition can be looked for. Permanent residence for some years in a favourable district is the only really curative treatment; but this may with benefit be combined with a summer visit to one of the hot sulphur springs; or to Ems, Soden, or any of the saline muriated and carbonated springs, suitable for the individual case. The opportunities for permanent residence in eligible climates are widening year by year.

Prophylaxis.—(i.) *Prophylactic measures between the attacks.*—No risks should be incurred by the chronic bronchitic patient. Sudden changes of temperature, as at sunset, or from walking out of heated rooms into the cool of the night, or into cold and damp buildings after exposure to the sun, cold winds, dampness of air and of soil, dusty localities and occupations, great variations in the amount and thickness of clothing, chill from damp underclothing after perspiration, and, almost above all, inactivity of the liver should be sedulously guarded against. The merely passive avoidance of obvious dangers is, however, a lame policy; we should be prepared for those which are apt to fall upon us unawares. Bracing resorts help us in this by toning up the nerves and tightening the membrane. A great deal can be done by the patients themselves in utilising the opportunities afforded by protective climates for the combined development of muscular energy and of respiratory activity. It is to be observed that vesicular emphysema is almost entirely a passive change, not brought about directly by voluntary expansion of the chest. In my opinion, systematic and graduated respiratory exercises, though they may stretch, tend to strengthen rather than to weaken the elastic fibre; and since they are based upon the performance of effective expirations, they would appreciably relieve the passive emphysematous distension. Much of the hepatic and of the local bronchial congestion will also be corrected by the greater activity of circulation thus initiated; and increased oxygenation will promote the growth of a less irritable and delicate epithelium.

The same tonic system can profitably be applied to the skin by means of a well-planned course of rubbing, bathing, and douching. All these measures need long perseverance before their beneficial effects can be fully secured; but their sedulous employment will bring with it an almost assured reward.

(ii.) *Prophylaxis in early bronchial delicacy.*—Yet more important is the subject of prophylaxis in infancy and childhood. The bronchial tubes are apt to suffer early in life; worst of all is the mischief arising from a severe attack of whooping-cough. Inherited family tendencies may in some children point also to a future liability to bronchitis. Moreover, in the case of all children, and especially of town-bred children, we have to deal with the liability induced by climate. All infants in this country, but in special and varying degrees the offspring of delicate, asthmatic, bronchitic, and gouty parents, stand in need of the help of preventive measures. If this were thoroughly understood and our practice regulated accordingly, a vast saving of life and health would be secured. The prophylactic plan suggested can be summed up in one word. It is a "hardening" plan carried out with vigilance and discretion; its essentials lie in the management of respiration and atmosphere, of temperature, of clothing, and of the skin.

Respiration and the atmosphere.—It is not sufficiently recognised that the bronchial tubes and lungs are constructed for the air we live in, and conversely. Specially strong is the prejudice against night air, which in itself is exceedingly beneficial. The innocuousness, for the bronchial membrane, of the higher temperatures of atmospheric air needs no demonstration; the innocuousness of extremely cold air, though it is not usually brought home to us, is evidenced by the ease and comfort with which respiration is carried on in arctic temperatures. Much of the objection to night air is generally directed against the dampness of it; but moisture need not in itself be detrimental; indeed, as we have seen, it is often used as a remedy. Nevertheless any of the normal atmospheric peculiarities may cease to be beneficial and may be turned into a source of irritation by a systematic substitution of artificial atmospheres for that provided by nature.

The great prophylactic method is to see that infants and children live and sleep in the open air as much as possible during the day, and enjoy as much free ventilation from the outer air at night as may be compatible with prudence. The full measure of this fresh-air treatment may be attained by degrees only; but it should be persistently aimed at. In towns this rule is of much greater importance than in the country. The extraordinary amount of health enjoyed in London by the children of the poor, in spite of so much that is depressing, is in great measure to be explained by the out-door life they are obliged to lead in their dark streets and alleys.

The skin and temperature.—More serious still than the neglected training of the aerial mucous membrane is the neglected education of the heat-generating function in relation to the skin. An excessive amount of clothing by day and by night, with wraps round the neck and wool next the skin, excludes too completely the oscillations of the outer temperature which should act as stimuli to the cutaneous surface. Moreover, the constant moist heat which is thus maintained tends to make the skin delicate and to depress its power of reaction. Flannel underwear is the best and safest for subjects too feeble to keep up their body heat; and it is an invaluable provision against unusual variations in the atmospheric temperature or in cutaneous action, as in athletics, campaigning, rapid journeys through extremes of climate; but its constant use is not part of the systematic training of the skin. In healthy children and adults it is as a rule superfluous at night, although indispensable for children suffering from rickets, restlessness in sleep, or enuresis. When it is worn during the day the outer garments should be made proportionately lighter. To pile up heavy outer clothing over thick flannel undergarments is bad hygiene, and cannot fail to weaken growing children.

Hygienic treatment of the skin.—Active means of promoting a vigorous habit of the skin should not be neglected. Massage is almost superfluous in children, whose life is perpetual movement. The chief indication is the sponge bath or the douche and rubbing. Few children will fail to take kindly to the cold bath if trained with sufficient tact to its use. As a rule, there will be no difficulty in obtaining the glow of cutaneous reaction after the bath, by friction with a coarse towel. In some constitutions the cutaneous circulation is slow to recover itself, and some special modification of the bath is called for. An essential precaution is the application of plenty of warmth immediately before and immediately after the cold sponging. The child may be placed for a minute or two into a warm bath, transferred to another bath for cold or tepid sponging, and again put into the warm bath for an equally short time, before towelling. An alternative, and in some ways a better method is to sponge the surface rapidly with warm water whilst the child is standing in a warm foot-bath. After the cold sponging he is to stand again in hot water whilst the body is being rubbed dry. The latter method is extremely simple and very effectual. Adults also who otherwise might be debarred from the boon of the cold bath are in this way enabled to resort to it with perfect safety and with enjoyment. In nurseries a bright fire should be burning before the cold morning baths are given. The daily cold affusion is of the greatest value as a direct protective against "catching cold"; and its systematic use must be reckoned among the most powerful helps in training a habit of resistance, and of ultimate indifference to all ordinary bronchial or cutaneous impressions, in those whom inherent debility or inherited predisposition would otherwise have exposed to ever-recurring risks of bronchitis.

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BRONCHIECTASIS

Bronchial dilatation, when slight or limited to one tube, may escape clinical observation; usually it involves several of the cartilaginous bronchi, and then gives rise to unmistakable symptoms, constituting a clinical disease to which the name "bronchiectasis" is appropriated.

The name "bronchiectasis" is also in common use in descriptive pathology; but the affection is far from presenting in its anatomy that uniformity which we recognise in its clinical symptoms and signs. Walshe says: "The conditions of disease to which dilated bronchi may form an adjunct are: bronchitis, acute and chronic; emphysema; constriction of the tubes themselves; acute and chronic pneumonia; cirrhosis of the lung; phthisis, cancer, and chronic pleurisy with contracted side." So great are the differences between the various pulmonary lesions thus apt to be associated with it, that we regard bronchiectasis as a structural change which may result from a variety of morbid processes, rather than as a definite and independent pathological product.

Clinically speaking, bronchiectasis is a chronic affection implicating bronchi of good size; to this it is that the literature of the subject almost exclusively refers, and that the present article is chiefly devoted. In its anatomical sense the name is more comprehensive: it applies to the secondary and slighter dilatations as well as to those which are sometimes described as primary; and it belongs with equal right to air-tubes of all sizes. In a complete clinical nomenclature the affection as it occurs in the bronchioles should not be left out, and we should recognise a bronchiolar dilatation or bronchiolectasis, as well as a bronchiectasis; both varieties occur independently, and are clinically important. With a view to mark the distinction between them, which has not been much dwelt upon, they will be described under separate headings.

In another group, that of the secondary bronchiectases, tubes of intermediate size are commonly involved. This variety does not possess the same clinical interest, and our references to it will be incidental only.

I. CAPILLARY BRONCHIECTASIS, OR BRONCHIOLECTASIS (including the so-called "Acute bronchiectasis").—This type of bronchial dilatation stands out in clinical contrast with the ordinary variety, while its distinct pathological features throw light upon the pathology of bronchiectasis in general. In its most striking form it occurs in *children* as an acute process; and as such it was described by Andral, Rilliet and Barthez, and others as "acute bronchiectasis." The post-mortem recognition of a dilatation of the small tubes in connection with certain clinical symptoms noted during life led observers to infer that in other cases also, which presented the same symptoms but ultimately ended in recovery, the same lesions had existed without proving fatal; and it is upon this assumption, which is probable enough but not capable of demonstration, that rests the current belief that children may completely recover from acute bronchiectasis. Granting that recovery may be possible, it may in a proportion of the cases be but partial; and in these the dilatations persisting in some portions of the lung may lapse in the course of years into the common bronchiectasis of the larger tubes.

In the *adult* localised dilatations of bronchioles are frequent in chronic bronchial catarrh, and may follow an acute purulent bronchitis; but, so far as I have observed, they do not extend to the whole lung; and their accompaniment is not atelectasis, but chiefly emphysema. Their value is rather that of a complication than of a disease. Since, however, their symptoms do not differ from those of a catarrhal bronchitis, and do not add largely to the fatality of the latter, it would be hard to say that acute dilatation of the smaller tubes may not occur more often in the adult, and more often be the origin of true bronchiectasis than is commonly thought.

In its chronic form bronchiolar dilatation is relatively of small importance. It is a local lesion secondary to the respiratory inactivity of a pulmonary district disabled by bronchial obstruction, or hampered by adhesions; in short, to imperfect expansion of the lung with resulting accumulation of mucus. Its most common seat is the apex of the lung in phthisis, where, although an old vomica may have undergone considerable contraction, the collapsed alveolar substance in its vicinity had failed, owing to surrounding fibrous changes, to expand again completely. Small thin-walled bronchi, distended with clear or purulent mucus, may often be seen in these partially aerated and inactive remains of healthy lung tissue.

The same change may, however, be met with in an opposite association, in emphysema due to chronic bronchial catarrh. Where the emphysema tends to become bullous the dilated bronchioles may take a share in the formation of the bullæ, and occasionally perhaps in the production of pneumothorax.

The acute form is of much greater clinical interest. In the majority of cases its mode of origin is tolerably obvious from the clinical history. It is illustrated in the cases collected in Dr. Walter Carr's paper on "Bronchiectasis in Young Children"; and it is well displayed in the drawings reproduced, by kind permission of the editors of the *St. Thomas's Hospital Reports*, from Dr. Sharkey's paper on "Acute Bronchiectasis." In children the lesion is essentially the result of an acute catarrhal bronchitis and peribronchitis, with multiple and widely-diffused secondary collapse. As immediate factors, the bronchitis of measles and of whooping-cough probably contributes more cases than any other kind. The course of the disease and its clinical features are not very distinctive, as may be gathered from the brief account given by Dr. Sharkey of his two cases. The cases were not diagnosed as bronchiectasis during life.

In the first patient, æt. 2 (Fig. 2), there was no previous record of illness except measles. The lungs after death were pale and curiously dotted with black pigment spots, hard to the touch. The centre of each of these was occupied by a small bronchus. The bronchioles were everywhere dilated, and scattered here and there were what appeared to be small miliary tubercles; but the other organs were free from tubercle. Microscopically acute peribronchitis was found, accompanied with extreme bronchiectasis, and a little, but very little, emphysema. No genuine tubercles were seen.

The other patient, a child aged 4, was under observation from May 7th to June 10th, 1893. He had always been healthy till cough began, two months prior to admission. Since then he had spat up thick phlegm, and had vomited three or four times a day; but he was able to attend school until admission. At that time he presented a dusky flush, rapid breathing, no marked dulness on percussion, no tubular breathing, but crepitations over the whole of both lungs. The pulse-rate was 136; the temperature 102·6°; the respirations 44 per minute. The temperature gradually fell, but on June 3rd subcutaneous emphysema occurred; otherwise no material change took place till death. The lungs were found bulky, their surfaces thickly strewn with soft, round, transparent, bladder-like elevations, the cavities of which were perfectly smooth, and either empty or full of frothy mucus. Scattered through the lungs these small cavities, the largest of which was about the size of a pea, gave a worm-eaten appearance. The larger tubes were not perceptibly dilated or diseased, but there were numerous patches of broncho-pneumonia of small size, and here and there some collapse; but no tubercle. The microscope detected widespread acute bronchitis, peribronchitis, broncho-pneumonia and pulmonary collapse. The bronchioles were extremely dilated, and there was also considerable emphysema.

A diagnosis of dilatation of the bronchioles cannot be made with any certainty, even in children; or even when, as in these cases, the change is general and extreme. At most its presence can be guessed at. Neither percussion nor auscultation can fasten upon any trustworthy sign, and the character of the expectoration does not differentiate the affection from severe catarrh. In its localised occurrence in the adult dilatation of the small tubes is still less capable of recognition.

Prognosis.—The acute puerile form, as shown by the cases narrated, is sometimes the result of a catarrh so severe as to be in itself fatal. In other cases, perhaps, the bronchiolar affection may be limited to a portion of the lung; and the catarrh getting well, the small tubes may lose

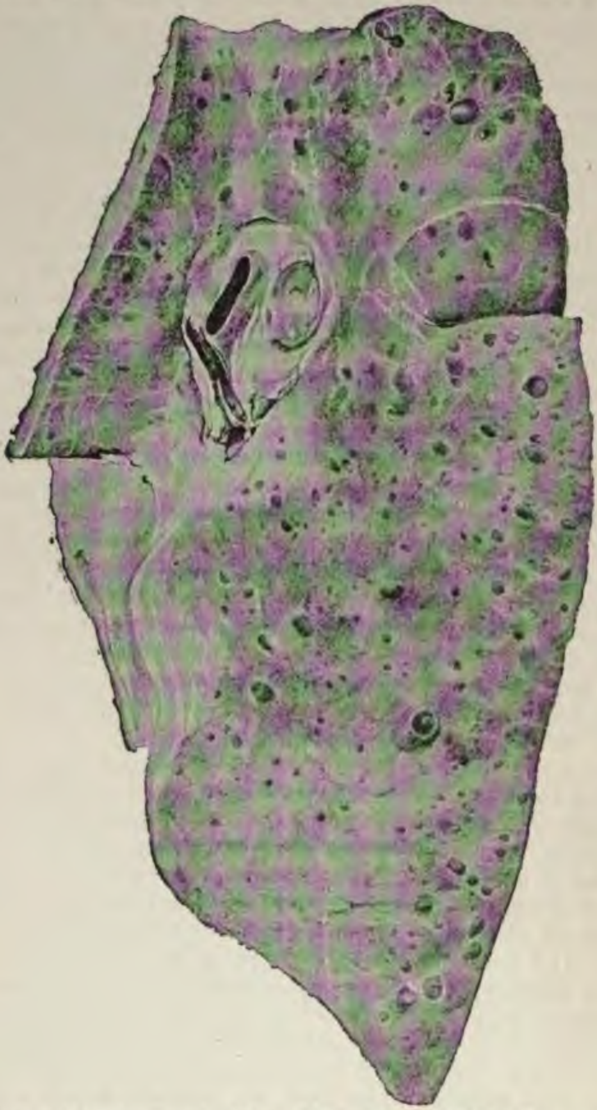

FIG. 2.—Acute bronchiectasis. Reproduced from Dr. Sharkey's Plate I. The figure represents the external surface of the lung, which is seen to be dotted with vesicles. In the fresh state they projected boldly on the pleural surface.

their dilatation. That this does occur is the view generally held; but, so long as a diagnosis of capillary bronchiectasis by physical signs is impossible, this must remain an unproved though a plausible opinion. Considerable likelihood has recently been added to it by the successful results obtained in cases of bronchiectasis in the adult.

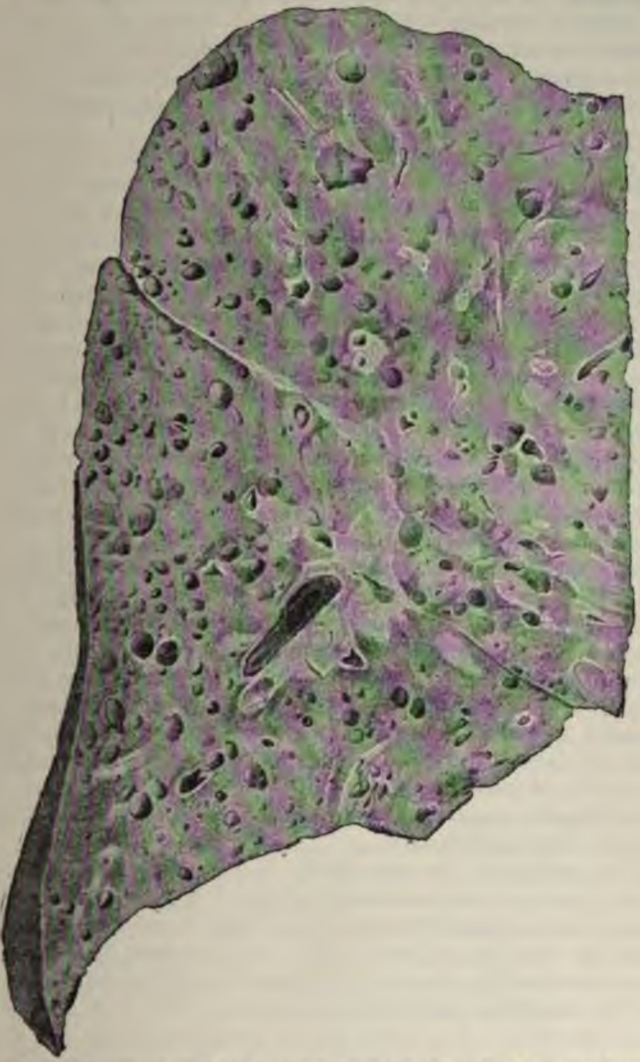

FIG. 3.—Acute bronchiectasis. Reproduced from Dr. Sharkey's Plate II. A vertical section of the same lung as in Fig. 2, showing dilated bronchioles distributed over the whole surface.

The treatment of an affection incapable of diagnosis cannot be laid down with any definiteness. In the chronic form none may be needed, the general symptoms being themselves chronic and sometimes unimportant. In the acute affection the presence of the bronchitis and of the catarrh supplies all the important indications; and these are sufficiently dealt with elsewhere. The great object in bronchitis being to prevent stagnation in the bronchioles, of which this form of dilatation is one of the results; the treatment of both diseases is practically identical.

II. BRONCHIECTASIS.—**Morbid anatomy.**—Since the time of Laennec, to whom we owe the first anatomical and clinical account of the disease, three main varieties of dilatation have usually been described: (i.) the regular or cylindrical, (ii.) the fusiform, and (iii.) the globular or sacculated. A modification of the *globular* is the *bead-like* variety, in which a tube may present at intervals a normal calibre between successive distensions. *Saccular* dilatations, with that exception, are terminal. The *cylindrical* expansions, on the contrary, affect the tubes as they pass towards the periphery. If a further dilatation should occur at their peripheral end, and cause the latter to become bulbous, the *fusiform* variety is brought about.

The largest and most extensive bronchiectases are found in more or less fibrotic lungs. Dilatations occurring in emphysematous surroundings are usually either fusiform or bulbar dilatations of single tubes, or cylindrical expansions of sets of smaller bronchial tubes which may be filled with catarrhal secretion.

Congenital bronchiectasis, the varieties of which constitute a distinct group, may be regarded as a malformation, or as resulting from some intra-uterine disease, perhaps syphilis. Usually one lung only is affected, and may present a large cyst with a central space branching into a peripheral set of intercommunicating secondary and tertiary cysts, with serous contents. Instances of this kind have been described by Grawitz, by Kessler, by Meyer, and by Fränkel. In another variety described by Grawitz, numerous separate cysts are formed on the bronchi of the third and fourth order; some of them communicating with the bronchial lumen, others being entirely closed. Goitre was found associated with this malformation.

In the atelectatic bronchiectasis described by Heller there is an abnormal growth of the bronchial cartilages, together with remnants of unexpanded, non-pigmented fœtal lung tissue; and the epithelial lining is not of the columnar ciliated, but of the pavement type. Cases have also been described by Gairdner, by Francke, by Herxheimer, and others.

Lastly, congenital bronchiectasis may be due to a *dermoid growth* within a bronchus. An almost unique specimen, now in the Museum of St. George's Hospital, was exhibited by Dr. Cyril Ogle before the Pathological Society on March 2nd, 1897. The patient, a male aged twenty-eight, had suffered intermittently for five years with cough and hæmoptysis, and ultimately died from profuse hæmorrhage, after a period of hectic temperature, fetid expectoration, and physical signs suggesting empyema or bronchiectasis; both of which were found after death. The dermoid mass, consisting of cheesy sebaceous material which contained loose hair and a tooth, was attached to the internal surface of a primary division of the right bronchus; this division was much dilated, and continuous with a large cavity in the substance of the lower lobe.[1]

Situation of the dilatation.—Bronchiectasis may be limited to one lung. Lebert found in fifty-four autopsies an affection of a single lung in 52 per cent; and of both lungs in 48 per cent. Even when restricted to one lung the dilatations are usually multiple, and they may occur in any situation. Lebert's figures are interesting in this respect. In his twenty-eight cases of unilateral bronchiectasis, six (21 per cent) presented an affection of the upper lobe; one (3 per cent) of the middle lobe alone; nine (32 per cent) of the middle and lower lobes; and twelve (42 per cent) of the whole lung. The view held by Laennec, Stokes, and others, that the apex is the commonest site of bronchiectasis, may have arisen from an imperfect distinction between tuberculous and bronchiectatic lesions. In a further series of fifty-five cases, observed only during life, fifteen (29 per cent) presented bilateral signs. In the remaining group of unilateral cases the upper lobe suffered in six (55 per cent); the lower lobe in fifteen (37 per cent); and the entire lung in fifteen (37 per cent). It would thus appear that in practically half the cases the affection is unilateral.

The distinction between tuberculous cavities and simple dilatation occurring at the apex never presents any difficulty, except in chronic cases of phthisis where a vomica has emptied itself of all caseous matter, and presents a smooth and relatively dry surface. This latter condition was described by the writer in the Goulstonian Lectures for 1882. Close inspection will show: (i.) that the bronchus opens into the cavity too abruptly for bronchiectasis; (ii.) that the bronchial membrane can only be followed over a small surface immediately adjoining the orifice of the bronchus; and (iii.) that the wall of the cavity presents none of that sculptural detail which identifies the original structure of a bronchus even in extreme dilatation.

In a section through a much-contracted fibrotic apex bronchi of normal size may appear to be enlarged, owing to the disproportion between the atrophied lobe and its larger air-tubes, which are shortened and slightly widened by its retraction. Moreover, it should be borne in mind that destructive tuberculous lesions of any part of the lung render a progressive dilatation of tubes belonging to the same bronchial set improbable, if not mechanically impossible; the damaged portion of the bronchial tree having become leaky, as it were, and unlikely to sustain much pressure. In the softening of phthisis the tendency is to an early ulceration and destruction of the tubes; and, as stated in the Goulstonian Lectures on Pulmonary Cavities, although during the progress of excavation the blood-vessels may persist for a long time in the trabeculæ, the bronchi—even those of large size—which traverse the diseased region are laid open and removed by ulceration at an early stage.

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[1] A similar hairy mass, growing in the upper lobe of the left lung, in communication with the bronchus, is depicted in Albers' Atlas. It was removed from the body of a woman æt. twenty-eight, who had been subject to pulmonary catarrh from childhood, and had for at least twelve years observed the presence of hairs in her expectoration. She died of exhaustion after hectic fever, dropsy, colliquative diarrhœa, and pulmonary congestion.

In the emphysematous tissue surrounding very chronic and practically healed lesions of the apex it is not uncommon to find unimportant dilatations of the peripheral air-tubes due to a rarefaction of the lung substance; these, however, are hardly to be dignified with the name bronchiectasis.

The changes in the mucous membrane and in the outer bronchial coats.—So long as the mucous membrane escapes destruction—and it is remarkable how long it will remain intact—it presents the signs of catarrh. In its later stages, however, it loses the velvety look, and assumes rather a smooth and shiny appearance consistent with atrophy of the epithelial layer. Most probably in all cases the atrophic changes prevail; although in some they may be limited to the internal coat, the adventitia taking on an inflammatory action which explains the thickening described as the alternative change. In Walshe's words, "The walls of such dilated portions of tube, commonly thick, and exhibiting the several characters assigned to tubes affected with chronic bronchitis, are, on the contrary, in rare instances thin and almost transparent." In general the instances of thickened bronchial membrane are those in which the inflammatory process extends around the dilated tubes into the pulmonary and interstitial tissue; whilst the bronchiectases with thin walls belong to the emphysematous group.

The condition of the mucous membrane differs much in the several varieties and stages of the disease; it is swollen and congested in the acute form (as in the acute cases of childhood), and in those chronic cases which remain free from much accumulation; congested and atrophic in cases of an opposite process; and, lastly, sometimes ulcerated or even gangrenous[1] in the later stages of extensive retention, when septic inflammation has supervened.

Hanot and Gilbert have connected the occurrence of hæmoptysis in bronchiectasis with the marked alterations described by them in the blood-vessels, which may form in the submucous tissue an extensive cavernous network, interspersed with numerous minute aneurysms.

According to Professor Hamilton the basement membrane of the original bronchus seldom gives way, but becomes stretched and attenuated. "On the basement membrane stratified columnar epithelium in a wonderful state of preservation may sometimes be found."

The changes in the surrounding pulmonary tissue.—As stated by Walshe, "The surrounding tissue is either slightly condensed by pressure, hardened by chronic pneumonia, rarefied by emphysema, or perfectly natural." Ulceration occurring in a sacculation is prone to set up fatal pulmonary gangrene. This was observed in twelve cases out of twenty-four by Rapp; in three out of forty by Barth; and in five out of fifty-four by Biermer. The gangrene, as in an isolated case mentioned by Lebert, may perforate a branch of the pulmonary artery. Perforation of the pleura would probably be less rare than it is but for the adhesions which so commonly exist and check the production of pneumothorax and of subcutaneous emphysema. Both these conditions have, however, been observed.

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[1] Marfan devotes a special chapter to "gangrene of the bronchi," which he regards as distinct from pulmonary gangrene and from putrid bronchitis.

Sir T. Grainger Stewart has described the process of absorption by which bands are left stretching across bronchiectatic cavities; or the latter may become multilocular, as often seen at the pulmonary base.

Inflammatory changes in the pulmonary tissue in the vicinity of the lesions are common. Acute pneumonia was recorded in twelve cases by Biermer, and in five by Lebert. Some inflammation also extends to the air-tubes in general. Hypertrophy of the bronchial cartilages, and a calcification of the walls of the dilated tubes—which in the bovine species is stated by Biermer not to be uncommon—have been described in isolated cases.

A cystic form of bronchial dilatation has sometimes been described (Biermer, Briquet); the cysts, which average the size of a walnut, being associated with a bronchial stenosis situated higher up. The contents may be serous, mucous, caseous, or even calcareous.

The secretion found in the dilated bronchi at different stages varies in its fœtor, and in the proportion of its fluid and of its solid constituents. Among the latter may be found: (*a*) recent mucus; (*b*) small casts, described by Dittrich and by Grainger Stewart, sometimes presenting epithelial flakes; (*c*) stale, opaque mucus undergoing granular and fatty degeneration; (*d*) micro-organisms of putrefaction (including sometimes sarcinæ and leptothrix pulmonalis, to which is due the purplish colour reaction of the bronchial casts on the addition of iodine, etc.), but no bacilli of tubercle. Occasionally the contents are blood-stained. Very frequently, though not always, crystals of the fatty acids and of cholesterine are found, especially in the fetid stage. Calculous concretions (Stokes, Dittrich) have also been observed.

Pathological changes in distant organs.—Various accidental complications have been described, such, for instance, as cancer, which Barth recognised in 8 out of 43 cases. The associated changes special to the disease are chiefly those connected with the obstructed circulation through the lungs: secondary dilatation of the right side of the heart, and venous congestions in the portal and in the systemic circuit. Valvular lesions may coexist, but do not appear to be traceable to the disease; pericardial adhesions sometimes occur as an extension of the pleuro-pulmonary fibrosis. The liver is almost always congested; it may present fatty change, and is sometimes lardaceous. Lardaceous degeneration also occurs in the kidney; and catarrhal nephritis has been recorded. Septic abscesses may be set up in various situations; one of their most common sites is the brain.

An *articular* affection, analogous to gonorrhœal synovitis, or to that sometimes following dysentery, has been described by Gerhardt in two cases of bronchiectasis, and is regarded by him as secondary to the bronchial trouble.

We should also mention the *skeletal* changes, not limited to this disease, described by P. Marie and by Souza-Leite under the name of Hypertrophic Pulmonary Osteo-arthropathy, and previously noted by Bamberger. In extreme cases there may occur, in addition to the usual clubbing of the finger-ends, an enlargement of the joint ends of the phalanges and metacarpals, of the long bones of the arm, and even of the vertebræ. Similar changes are also traced in the bones of the lower limb [*vide* vol. iii. p. 153].

Bamberger believes that the changes in bronchiectasis constitute a separate variety distinguished by the painful swelling of the epiphysis, and by the condensation occurring in its spongy substance as well as in its shell of hard bone.

General and clinical causation.—The insidious beginnings and the chronic course of bronchiectasis are not favourable to a study of its causes. Statistics of the disease at *various ages* can only deal with approximations. Lebert, in a series of 83 cases, found 47 per cent occurring before, and 53 per cent occurring after, the age of 40:

7	per cent	occurred	under the	age of	10		
8	,,	,,	,,	,,	20		
20	,,	,,	from the	age of	20	to	30
12	,,	,,	,,	,,	31	,,	40
18	,,	,,	,,	,,	41	,,	50
11	,,	,,	,,	,,	51	,,	60
24	,,	,,	,,	,,	61	,,	85

The congenital dilatations are exceedingly rare.

The male sex is more often affected than the female, according to Trojanowski and Bamberger; but other authors (Biermer and Willigk) have traced no difference. *Occupation* does not influence the production of the disease in any direct way, though it may act indirectly by setting up pulmonary and bronchial changes favouring a dilatation. Depressing circumstances of all kinds might also have an indirect effect.

Clinical antecedents.—We have no proof that the change ever arises spontaneously during extra-uterine life. In children we are able to trace its acute form to bronchitis. Fatal cases of this kind furnish us with the only direct evidence in favour of a definite causation from acute inflammatory disease; but clinical observations, although less conclusive, lend their support to the same view. When not traceable to an acute attack, dilatation is probably secondary to some chronic bronchial or pulmonary affection, and the precise time of its onset becomes difficult to determine.

As regards the immediate etiological factors Lebert's results are probably trustworthy. In a quarter of his series there had been previous emphysema; in another quarter an acute pleurisy or an acute pneumonia had preceded the disease; and in a large number the history was one of long-continued bronchitis with intercurrent acute attacks (Wilson Fox). Thus, bronchitis in all its forms, but especially when complicated with spasmodic cough, as in whooping-cough (Laennec) and in asthma (Hyde Salter), contributes a well-marked etiological group; pulmonary diseases, whether acute or chronic, rarefying or condensing, forming a second group; and pleuritic affections a third. A fourth group is that in which a temporary or permanent narrowing of a large bronchus, as by an aneurysm, has led to increased strain or to accumulations within its subdivisions.

The relation which the bronchial affection may bear to tuberculous disease has been much discussed. Some, including Rokitansky, have regarded the two diseases as almost incompatible, and as mutually protective. Nevertheless, true bronchiectasis may occur in the subjects of chronic tuberculous disease; for instance, at the base of a lung with an indurated apex. And, conversely, sufferers from chronic bronchiectasis may end in tuberculosis, though this is rare.

Wilson Fox suspected that the fibrotic induration around the tubes was probably tuberculous in its origin; the other tuberculous deposits in the same lungs having been slight and obsolescent: but this opinion does not appear to have had the support of any direct evidence.

Biermer, who quotes Trojanowski as reporting tuberculosis in 21 out of a series of 68 cases, could find only 3 in his own collection of cases. As pointed out by Wilson Fox, discrepancies of this magnitude can only be explained on the score of some confusion between tuberculous lesions and those due to bronchiectasis.

Pathological etiology.—The history of the subject is a record of hypotheses as varied as the associated intrathoracic conditions; but they may be briefly classified as attempting to identify the causation (1) with changes limited to the tubes themselves, (2) with changes in the pulmonary tissue, (3) with changes in the pleura, or lastly (4) with a combination of the bronchial, pulmonary, and pleural changes.

Some cases carry their own explanation: cicatricial stricture, lateral pressure from aneurysms or morbid growths, internal obstruction due to tumours, and particularly the impaction of foreign bodies are occasional causes of bronchiectasis; but those needing elucidation form a much larger group.

Laennec regarded the dilatation as due to an accumulation of mucus. Andral accepted this view only for the bead-like form, and attributed the other dilatations to a process of hypertrophy analogous to that of other hollow organs; this was also in part the view of Louis. Rokitansky, and subsequently Hasse, assumed a stenosis of the larger and an obliteration of the smaller bronchi, with compensatory dilatations elsewhere. Stokes and Williams traced the production of dilatation, under stress of cough or of accumulating secretion, to impairment of elasticity and of muscular contractility by inflammation. Atrophy of the bronchial muscles has been described by Bamberger, by Trojanowski, by Lebert. and as a primary and probably constitutional defect, by Sir T. Grainger Stewart. Lebert also suggested that dilatation might be due to atony dependent upon defective innervation. Various other pathologists (Beau, Maissiat, and Mendelssohn) have insisted on the share taken by cough in the production of dilatation.

Wilson Fox considered all forms, except those secondary to a constriction, as essentially inflammatory in origin; the loss of elasticity and muscular contractility of the tubes themselves being the only essential changes, and sometimes the only changes found; whilst on the other hand the dilatation would be favoured by the coexistence of a pneumonia, or of a broncho-pneumonia, or by pulmonary collapse or tuberculous indurations in the surrounding tissue. The acute bronchiectasis of infantile bronchitis he regarded as due to cough pressure rather than to any inspiratory mechanism, or to any indirect effect of collateral collapse; but the proofs upon which he based this view were not fully stated by him. As an explanation of the infrequency of bronchiectasis, in spite of the great frequency of bronchitis, Wilson Fox alleged that the dilatation is readily recovered from in children; and that in adults chronic bronchitis tends to hypertrophy rather than to weakness of the muscular fibres, in contrast with its action upon the pulmonary parenchyma.

It is noteworthy that Biermer traces as many as a quarter of the aggregate cases to acute pneumonia. The strict priority of the pneumonia is in many cases difficult to establish, and therefore open to some doubt.

Biermer is also a believer in the influence of pleural adhesions, which, according to Wilson Fox, are more easily explained as a secondary process. A compression of the lung by fluid was regarded by Buhl as most likely to lead eventually to bronchial dilatation. In this connection it may be pointed out that in simple pulmonary collapse no dilatation can occur in previously healthy tubes, so long as they receive evenly from all sides the strong support of carnified tissues; and that on the other hand the appearances of dilatation are very apt to be simulated by the shortening and retraction of tubes within a collapsed portion of the lung.

The explanation given by Sir Dominic Corrigan of the mechanism of the dilatation in cirrhosis of the lung has become classical. Owing to the rigid connection of the surface of the fibrosed lung with the chest wall, not only will the spontaneous shrinking of the fibrous tissue lessen the distance between the chest wall and the bronchial wall, but every inspiratory effort of the former will take effect in dilating the cavity of the bronchus. Corrigan's theory was subsequently adopted almost unaltered by Rokitansky and by Lebert. The latter based the etiology, at least in fibrotic cases, on some antecedent *pleuritis profunda* setting up a proliferative irritation in the pleuro-pulmonary connective tissue.

Dr. David Drummond has favoured me with the following statement of his views on the production of bronchiectasis from *pleuro-bronchitis*:—

"The common form begins as an acute bronchitis and pleurisy, to which the name pleuro-bronchitis is fairly applicable. The process leads early to blocking of some of the larger tubes by hypertrophic thickening of the mucous membrane, and in consequence to collapse of lung and diffuse broncho-pneumonia. This form of pleurisy is essentially progressive. The fluid becoming absorbed, fibrous thickening of the pleura sets in. Tube after tube becomes blocked and subsequently dilated from pent-up discharge, which in time bursts away. After death the tubes first attacked are found surrounded by fibrous tissue; but those affected later are devoid of fibrous tissue, and only nuclei and collapsed lung can be found in their vicinity, showing that the fibrous tissue is developed after the dilatation of the tubes."

Most of these hypotheses are summed up and criticised by Sir T. Grainger Stewart and Dr. Gibson under the headings of:—(1) Direct pressure of stagnating secretion—a mechanism in which they do not believe; (2) Concentrated air pressure, as in cough (Reynaud, Williams)—an explanation which they regard as inadequate apart from another factor, that of an essential debility of the bronchial wall; (3) Extra-bronchial traction—which they recognise only in cases of pulmonary cirrhosis, and not in all of these, since in as many as 20 per cent Bastian found no dilatation; (4) Inflammation of the bronchial wall causing loss of elasticity, of contractility, and of ciliary movement (Stokes)—a view which, according to them, leaves unexplained the infrequency of bronchiectasis in spite of the great prevalence of bronchitis; (5) Dilatation as a result of defective innervation and loss of tone, as alleged by Lebert, an origin which they regard as unproved; (6) Lastly, Sir T. Grainger Stewart's own hypothesis, first published in 1867; which refers the origin of a large proportion of the cases to a constitutional, or possibly, as held by Leroy, to an hereditary weakness, a "primary atrophy" of the bronchial wall, unfitting the bronchi for stress even within the physiological limits of powerful inspiratory efforts, of cough, and of violent exercise. Once originated in an insidious manner, this *primary bronchiectasis* progresses to the fully developed forms with the well-known symptoms.

In another large group—that of the *secondary bronchiectases*, including a *general* and a *local* variety of dilatation—the same authors recognise among the determining causes the influence of pertussis, of capillary bronchitis, of bronchial stenosis or impaction, of pulmonary cirrhosis. Most of the explanations hitherto attempted have, according to them, been limited to these secondary varieties. Here again individual delicacy or inflammatory impairment of the contractility or of the elasticity of the bronchi may be frequent factors in the result.

The writer's views.—The first essential for a comprehensive theory of bronchiectasis is a sufficiently broad basis. There is one feature which is common to all; namely, the faulty distribution of space between the air-tubes and the pulmonary tissue. In health the intrathoracic space is suitably distributed between its several contents; the functions of which are regulated for the avoidance of undue stress on any one of them. Any excessive stress ultimately finds out the least resistant tissue, and this is most often the pulmonary tissue. Why, in exceptional instances of sustained intrapulmonary pressure, the bronchi should suffer rather than the pulmonary tissue, has never been explained. The possibility that disorderly nutritive changes may occur at an early period of development must be borne in mind; and hereafter vascular disease may be found to take as leading a part in the production of bronchiectasis as it does in that of emphysema. On the whole, however, there is little support for the view that bronchiectasis is a progressive deterioration due to an innate local delicacy, independently of disease.

In disease, mechanical factors arise which are entirely foreign to the natural play of the organ, and which do not necessarily seek out the weakest part. To these belong, within the tubes themselves, an accumulation of mucus and the antecedent or the resulting degenerative changes in the bronchial wall.

A second influence is that of changes induced in the lung tissue. In a rather large proportion of cases bronchiectasis is accompanied by more or less emphysema. Much of this is clearly a result rather than a cause, since the ordinary vesicular emphysema does not carry with it any accessory bronchiectasis.

Another frequent accompaniment of bronchiectasis is pulmonary collapse. When occurring unevenly, at one side of a bronchial tube, this may act as one of the agents of dilatation. Not only in advanced bronchiectasis do we often observe a proportionate amount of condensing fibrosis of the lung, but in any recent dilatation, such as that witnessed in the infant after bronchitis or whooping-cough, the incipient bronchial bulgings occur side by side with considerable lobular collapse.

A further set of structural changes contributing, in a large proportion of the cases, to faulty allotment in space, are those of the pulmonary stroma, which includes the subpleural, the perilobular, and the interlobular systems.

As to the *general mechanism* of the dilatation we must again look for some elementary factor common to all varieties; and this we find in "obstruction," understood in the broadest sense of the word.

In the alimentary tract and in most animal tubes the obstruction is invariably situated forward, beyond the dilating segment In the bronchial tract no such local restriction obtains. Neither is the nature of the obstruction necessarily limited to stenosis or to impaction. Owing to the alternating direction of the respiratory air-currents, an obstruction may lead to dilatation either on its proximal or on its distal side. Again, the dilating force is not usually, as in other tubes, the pressure of an accumulation within the dilating bronchus. This mechanism may occur in the bronchial system: an instance in point is the thin-walled sacculation, completely filled with stiff gelatinous mucus, sometimes found beyond a bronchial stenosis. But much more often the obstruction has its seat on the distal side of the dilatation and is not a bronchial stenosis, but a terminal occlusion of a respiratory district of the lung; and the dilating force, far from being exclusively due to the pressure of an internal accumulation, is then applied to the outside of the tube; it is an aspirating, not a forcing pressure.

If we bear these elementary data in mind we shall find that the details of the problem work out. Thus, whereas in the normal state each pulmonary constituent preserves its relative position and its allotted space, the local failure of any individual constituent to perform its respiratory function would interfere with the perfect adjustment of other parts during the phases of respiration. How readily bronchiectasis might result from this disturbance will be seen from a consideration of the forces which normally protect the weaker non-cartilaginous tubes against the dilating influences of atmospheric pressure. The elasticity proper to the inflated pulmonary tissue through which they pass tends to widen them; but this tendency is counteracted by the inspiratory elongation of the lung, and probably never goes farther in health than to ensure their patency, thus acting in the depth of the lung in lieu of a cartilaginous armature. On the other hand, both during inspiration and during expiration, the small tubes receive lateral support from their closely fitting environments. Let this support be withdrawn at any one spot by the persistent inspiratory inactivity of one of the adjacent lobules, even though this were merely a delay in the fulfilment of inspiratory inflation, then the imperfectly resisted intrabronchial pressure would gradually bulge out the yielding wall into the space rendered available, and thus establish the first stage of a progressive dilatation. Or, to put the matter more clearly, the inspiratory traction made by the chest wall, if it should fail to expand an obstructed lobule, might be transmitted to the delicate air-tube adjoining the latter, and might dilate it.

Owing to the solidarity existing between all parts of the lung, this *encroachment of bronchial space into the vacated pulmonary space* may occur at a distance from the original collapse. The same mechanism might therefore be concerned in some measure in the production of almost every variety of bronchiectasis. Its more strictly local operation is probably alone concerned in the early stages of the affection when the pulmonary tissue is still free from induration. In some instances bronchiectasis remains permanently uncomplicated with any pulmonary fibrosis, or with any peribronchial thickening. It is in these cases that the bronchial membrane preserves its delicate and transparent thinness. The plug of semi-gelatinous mucus which sometimes fills simple dilatations of this kind in the midst of soft spongy lung tissue suggests the idea that the mucus itself was originally the obstacle to the free inflation of the collateral lobules, whilst its accumulation eventually assisted in producing the distension.

The progressive increase in the dilatation may conceivably be brought about by the various mechanisms assumed by the so-called inspiratory and expiratory hypotheses; although much that has been advanced in connection with them is lacking in strict proof. Thus:—

(i.) *The inspiratory hypothesis* of Laennec asserts that the abnormal inspiratory effort preceding cough throws damaging stress upon the weakened parietes of the bronchial tube. In the diagram (Fig. 4) which illustrates this supposed agency, if we imagine the shaded zone to remain unexpanded, the arrows would represent the inspiratory traction thus transferred from the alveolar to the bronchial walls.

The same explanation has been applied to the condition which may result from a proximally situated stenosis, when the impeded removal of the products of catarrh from the terminal districts has led to an irregular lobular collapse with consequent disturbance of the balance of pressures.

(ii.) *The expiratory hypothesis* has also been pressed into the service of bronchiectasis as well as of emphysema. Were it not that one of the chief functions of man in earning his bread by manual labour is the performance of *muscular strain with closed glottis*, and that his organs are specially constructed for that purpose, the wonder would be that the prevalence of bronchiectasis and emphysema is not universal.

As a fact, nothing gives way within our visceral cavities under the high pressures due to muscular strain so long as every part is sound and works true. The extent to which we are dependent for this immunity upon a perfect distribution of pressures is illustrated by some of the delicate valvular membranes of the heart which could not perennially resist the stress to which they are exposed, were not the pressure exerted upon one of the two surfaces neutralised by equivalent pressure or support on the other. So must it be also with the delicate bronchial membranes. The range of pressures to which they are exposed is not so great, but their risk is multiplied by the number of their subordinate districts. A loss of the even balance between the intra- and the extra-bronchial pressure occasioned by imperfect inflation of any of the latter might in delicate and predisposed subjects cause the bronchial wall to yield, and to suffer progressive dilatation.

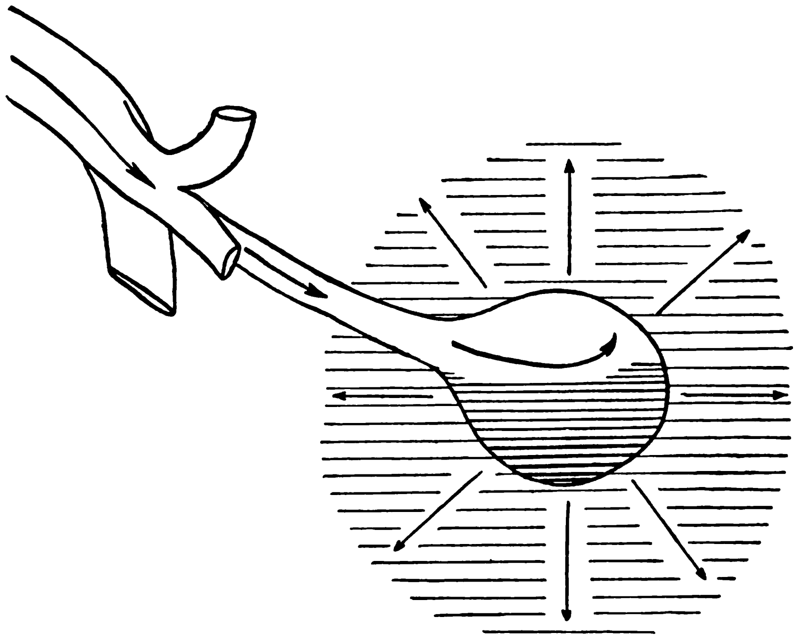

FIG. 4.—Illustrating the alleged mechanism of dilatation, according to the inspiratory theory.

Cough is a special instance of muscular stress; it is often complicated by the mechanical influence of the secretion which excites it. The diagram (Fig. 5) illustrating the mechanical theory of expiratory pressure will also serve to explain this point.

The cough which may be powerless to dislodge and evacuate the contents may yet propel some of them far enough to cut off the dilated chamber from the main bronchial channel. The moment represented is that of the explosive expiration, when the air accumulated under high pressure leaves the chest without any further hindrance. Alone in the dilated tube the pressure, indicated by the curved arrows, will remain at that moment nearly as high as during the period of closure of the glottis; and its dilating effect is but feebly counteracted by the released elasticity of the immediately surrounding lung tissue. Slowly, with the ensuing inspiration, the plug may be sucked in again; and this suction is the most likely explanation of the long-drawn, semi-musical, or croaking rhonchi and râles of bronchiectasis.

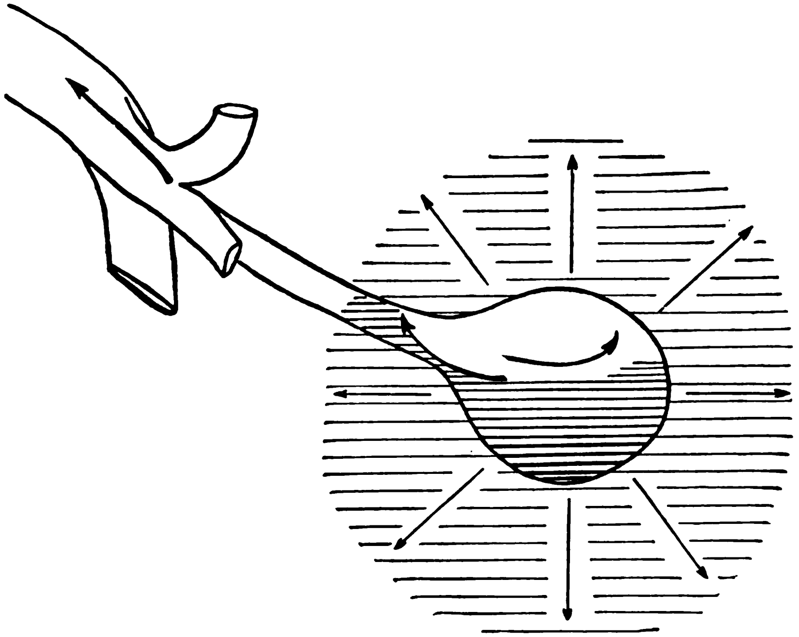

Fig. 5.—Illustrating some of the effects of cough in bronchiectasis. (From *Clin. Journal*, Feb. 1894.)

The practical results of a recurring valvular obstruction of this kind would be not only a continued fulness of the dilatation, whilst the surrounding tissues are being relieved of much of their air, but a maintenance within it of the highest air-pressure at the time when the air-pressure in its vicinity is at its minimum. Neither should we lose sight of the possible injection into the tributary bronchioles and lymphatics of some of the bronchiectatic contents.

Most cases may begin and progress after the mode suggested; but, except in fatal cases of bronchitis and whooping-cough in children where these etiological relations are well displayed, an opportunity of examining the lung at this stage is not often afforded; and ulterior changes of a very different kind usually obscure, more or less completely, the original mechanisms.

The influence of catarrh seems entitled to be regarded, as it has been by most writers since Laennec, as the chief and earliest etiological factor of bronchiectasis. The inflammatory softening and weakening of the bronchial wall, the changes in its muscular and fibrous coats, whether in the direction of atrophy or of overgrowth, are all possible accessory agents; but the special action of catarrh consists in the mechanical plugging of bronchioles. When a bronchiole becomes occluded the amount and the pressure of the air within its district are rapidly altered, and the balance of pressures will be disturbed to the special detriment of the tube from which the bronchiole sprang. If the pressure can be speedily readjusted by collateral expansion in the vicinity any strain or dilatation thus induced will be corrected. Failing this adjustment, however, the existing catarrh will aggravate the dilatation by a tendency to accumulation and by the impairment of the respiratory mechanisms of relief.

The successive obliteration by catarrh of many tributary bronchioles is probably the mode of extension of bronchiectasis. The greater the stretching of the dilated bronchial membrane and the accumulation within it, so much the greater will be the number of collateral bronchioles obliterated by stretching or by plugging, and so much the greater the extent of the resulting atelectasis.

A direct influence aiding the dilatation is that of any impairment of the muscular coat, whether in its structure, as in the atrophic fibrosis described by Lebert, or the simple atrophy of Grainger Stewart; or in its function, as in atony from defective innervation, or from insensitiveness of the mucous membrane.

Indirectly, the process of dilatation might be favoured, as in pertussis and in acute bronchitis, by the opposite condition of bronchial spasm, since this would lead to a narrowing and to a more ready plugging of the smaller tubes.

The influence of interstitial pneumonia and fibrosis.—In whatever way it may have arisen, a sacculation of a small bronchus is fatally exposed to an accumulation of secretion during periods of catarrh, and to irritation not only within its own terminal divisions, but probably, by overflow and by inhalation, in collateral lobules also. This is the beginning of an interstitial pneumonia, the ultimate result of which may be a conversion of the pulmonary substance into structureless fibrous tissue. The loss of expulsive power is progressive, and the shrinking of the chronically inflamed parenchyma favours the encroachment of the sacculation; whilst the implication of the lymphatics of the lobule causes an extension of the changes along the perilobular system. In this way the pulmonary degeneration is promoted along two lines, by intralobular and by perilobular agencies. How far it may extend will depend upon the varying ability of the remaining pulmonary tissue by its increased expansion to replace some of that which has atrophied. Dense adhesions would largely interfere with this compensatory process.

The influence of pleuro-pneumonic fibrosis.—The close relationship existing between the pleura and subjacent stroma and the lymphatic system of the lung explains the influence which agglutination of the pleural surface exercises on the course of the interstitial pneumonia, and on the etiology of bronchiectasis. Extensive pleuritic thickening at the base, with obliteration of the groove and agglutination of the surface of the diaphragm, cripples the lung. The respiratory function of the base is almost entirely lost, or can be carried on only by considerable mechanical effort on the part of the diaphragm, and of the inspiratory muscles; an effort which must tell on the pulmonary tissue as a constantly recurring and irritating traction. The lymphatic circulation may also be impeded. The result is usually a considerable shrinking of the side affected, and a compensatory hypertrophy of the sound lung, with great distension of that side of the thorax.

The process which has just been sketched is essentially that originally described by Corrigan under the name of "cirrhosis of the lung."

The influence of stenosis.—Dilatations are by no means the invariable result of bronchial stenosis. When a bronchiectasis occurs beyond the stenosis its mechanism is generally held to be analogous to that of emphysema from a partial obstruction of tubes, which allows a slow entrance, but unduly delays the escape of air. Syphilis, being a well-known cause of bronchial stricture, should be allotted a place among the recognised factors of bronchiectasis. It is not improbable that in some cases the occurrence of a late ulceration of the dilated tubes may be due to the same influence.

Hoffmann believes that sufficient attention has hardly been paid to the probably frequent origin of bronchiectasis from *inhalation of solid particles;* and he refers to the experiments of Cohn which show that dilatation occurs not beyond but at the seat of impaction, around the impacted foreign body.

Lichtheim's experiments go to prove that total closure of a bronchial tube leads within twenty-four hours to a complete atelectasis of the pulmonary district, with purulent accumulation within the tubes. After several weeks the latter become more or less dilated, the surrounding tissue being completely compressed by the distended bronchi, or expanded by collateral emphysema.

Beyond any valvular obstruction micro-organisms, which easily penetrate through the stenosis, may set up fermentation, and the secondary results of putrid decomposition will follow.

Symptoms.—The severity of the disease varies greatly in different individuals and at different stages in each. Its course and its symptoms are largely determined—(*a*) by the mechanical factors, such as induration or persisting elasticity of the surrounding tissue, position of the dilatation, its single or multiple character; (*b*) by constitutional factors special to the individual or to phases of his general health; and (*c*) by climatic and atmospheric factors, including not only temperature and humidity, but also purity of air, in the sense of relative freedom from septic germs.

It has already been stated that in exceptional instances bronchiectasis may be latent for some time after its commencement: in a few cases also there may be periods of quiescence during which it might pass unobserved. These are the milder forms, of a catarrhal and emphysematous type—non-indurative, non-septic, non-ulcerative, progressing but slowly, and compatible with relative longevity. All cases are liable to exacerbations in the symptoms, to occasional or periodical increase in the expectoration, to recurring intervals of fetidity of the sputum, and to intercurrent attacks of general bronchitis or catarrh.

Constitutional symptoms.—For long periods the flow of expectoration, sometimes even when fetid, may proceed without making any obvious impression upon the general nutrition or functions; but these are gradually involved as the diminution of respiratory surface and consequent loss of energy advance; and ultimately the system is contaminated by the septic matters inhaled, absorbed, and swallowed. The constitutional symptoms set in at different stages, and at first may not be permanent, but coincide with transient periods of fœtor of the sputum. In the worst forms these deteriorations are lasting. Sooner or later the pulse and respiration become permanently accelerated, and the temperature moderately hectic, or at the least remittent, with an evening rise to 101° or 102°, and in a few cases with associated night-sweats. Diarrhœa may be among the septic symptoms, and sometimes vomiting also. Vomiting as a mechanical result of cough is not so common in bronchial dilatation as in phthisis.

Failure of cardiac energy lies at the root of the final cachexia. In addition to the previous lividity and cyanosis œdema supervenes, and the patient becomes a bed-ridden invalid. At this stage, or prior to it, intercurrent albuminuria may be observed; or in association with lardaceous disease it may become permanent. Various complications may cut short the gradual process of exhaustion; low pneumonia, putrid bronchitis and gangrene, septicæmia or pyæmia, cardiac or renal disease, and cerebral abscess are among the most common. In the more favourable cases, especially when helped by the advantage of climate and treatment, the sufferers may live with their trouble for years, and die from other causes. Those who reach a relatively mature age are more and more exposed to catarrh and emphysema with their attendant symptoms, and the disease, whether directly or through its complications, is usually responsible for death.

Pulmonary symptoms.—Under this heading we must briefly review the changes in the respiratory function—the cough, the expectoration, and hæmoptysis.

Dyspnœa.—There is often a cardiac element in the dyspnœa observed in bronchiectasis. Much cardiac and nervous depression is induced at times by septic absorption from the bronchial tubes and through the breath, especially in ulcerative cases. As a rule, during the major part of the clinical history the dyspnœa is not excessive; but it varies much with the degree of emphysema or of fibrosis, and with the amount of intercurrent catarrh. In the ultimate stages dyspnœa becomes a prominent feature.

Cough.—A leading peculiarity of the cough of uncomplicated bronchiectasis is its intermittence. It would seem as though the sacculated membranes lost their sensitiveness, and that cough were excited only when the tide of accumulation reaches the level of some healthier part of the bronchial tubes. It is often observed that for long periods, during which a patient preserves the posture which acts as a protection, no cough is set up; but change of position will bring on severe spasmodic cough and profuse expectoration. The severity of the cough and its paroxysmal character are explained by the irritating quality of the secretion which has to be forwarded through the sensitive upper passages; and also by the difficulty, or impossibility in some cases, of complete relief on account of the position of the sacculations. Whereas a partial emptying of the surplus of the bronchial contents is comparatively easy, nothing short of an inversion of the patient could empty some of the ultimate dilatations, especially when surrounded by fibrous tissue. The creasote inhalation method introduced by Dr. Chaplin has demonstrated that the fœtor of this residual material is much in excess of that of the tidal output, a point which should be borne in mind as of the utmost importance in treatment.

Expectoration.—The sputum in bronchiectasis varies considerably in amount and in character. Sometimes it remains sweet and almost purely mucous for long periods; more usually it is muco-purulent throughout. In most cases it becomes fetid at times; or this may be the habitual condition. Very often, when ulceration has taken place or after severe paroxysmal cough, it is slightly blood-stained.

A third of a pint or half a pint is not an unusual daily quantity; but this amount is often much exceeded. The way in which the expectoration pours out of the mouth in some cases is almost distinctive, though the same peculiarity may be observed in phthisis. At intervals the expectoration may be much lessened or completely absent. Complications, especially bronchitis or pneumonia, commonly reduce the amount.

The sediment deposited by the expectoration, which may separate into an upper frothy mucus, and a lower puriform layer with an intervening watery layer, presents, besides bronchial epithelia, numerous pus-cells, granular débris, bacteria and vibriones, fatty acid crystals; and occasionally sarcinæ, leptothrix, Dittrich's plugs, and Charcot-Leyden crystals. The presence of elastic fibre would be a proof of ulceration. The fœtor is apt to be great, but it is occasionally more marked in the breath than in the sputum.

Hæmoptysis was among the symptoms described by Laennec. Walshe failed to observe hæmoptysis except in the presence of mitral disease or of tubercle. Lebert observed hæmoptysis, of varying degrees but decidedly more marked than that which belongs to ordinary pneumonia, in one-sixth of his cases. Biermer reports one case of fatal hæmorrhage in non-tuberculous ulcerative bronchiectasis. Wilson Fox, who quotes these authors, refers to it as not being rare. It may occur early and independently of any ulceration. On the whole, it is to be regarded rather as a frequent complication than as an invariable symptom.

The respiratory symptoms vary with the degree of the pulmonary atrophy. Among them are to be noted frequency of breathing and dyspnœa on exertion, and, in unilateral cases, inability to lie on the sound side.

Physical examination of the chest.—*Inspection.*—There is no distinctive chest shape peculiar to bronchiectasis; and the thorax does not present the characteristics of phthisis, even though one side may be much retracted. Whatever amount of flattening may be present locally, this is compensated elsewhere by active thoracic expansion. The immunity of the apex in the majority of cases and its compensatory expansion, coupled with the fulness of the neck, establish at first sight a distinction from the ordinary case of phthisis. Often, on the other hand, the deformity peculiar to emphysema may be more or less fully established. The unilateral character of the group of cases described by Corrigan as cirrhosis of the lung is usually made obvious by the cardiac displacement, and by the extreme disproportion between the size and the respiratory movements of the two sides of the chest. But in some unusual cases, owing to considerable encroachment of the sound lung across the middle line, the thorax on the side affected is much less collapsed than the lung which it contains. Cases of this kind are deceptive, and need, for an accurate determination of the size of the lung, a very careful percussion of the boundaries of the cardiac dulness. I have described a case of this sort. This cirrhosis of the lung without thoracic deformity is much less readily distinguished from phthisis or from chronic bronchitis than the usual form.

Among the bilateral cases the emphysematous variety is to be diagnosed from phthisis, on mere inspection, by the dusky and congested complexion, the prominent veins and deeply coloured lips, the high, deep, and broad chest, and the relatively good nutrition.

In the remaining groups the diagnosis may be assisted by a knowledge of the following points:—(*a*) A solitary bronchiectatic lesion is seldom localised at the apex; this is the customary site for the tuberculous lesion. (*b*) The supraclavicular area is usually not implicated in any dulness due to bronchiectasis; it is invariably implicated in the apex dulness of phthisis. (*c*) In phthisis, as pointed out by Stokes, consolidation precedes, excavation follows; in bronchiectasis this is otherwise. And again, extension of the excavation is peculiar to phthisis (Stokes), whilst a stationary size belongs to bronchial dilatation (Walshe). (*d*) The almost daily alternations between the signs of fulness and those of vacuity greatly help the diagnosis of sacculation. This peculiarity is usually absent or inconstant in excavating phthisis. (*e*) The normal site for tuberculous disease is the apex; it hardly ever involves the base. The site of predilection for bronchial dilatation is the base; but bronchiectasis also favours the middle and lower third of the back and may affect various other situations without any hitherto ascertained regularity of order; it is specially uncommon in the district of the vertical bronchi ascending to the apex. (*f*) It is unusual in phthisis for multiple excavations to form in the same lung with the intervention of sound pulmonary substance, except in the situations described in the Goulstonian Lectures for 1882, and by Dr. J. Kingston Fowler in his *Dictionary of Medicine*. Even these secondary deposits are commonly almost continuous with the upper zone of disease. In multiple bronchiectasis a truly sporadic arrangement is the rule. (*g*) Unilateral indurative tuberculous phthisis invariably excavates and condenses the apex first, even if later it should extend downwards. The fibroid change associated with bronchiectasis originates as a rule at the base and spreads upwards. (*h*) The displacement of the heart towards the diseased side of the chest in the usual cases of unilateral phthisis follows an oblique direction upwards; a horizontal displacement is exceptional and suggests some complicating basic pleural factor. In unilateral bronchiectasis the displacement is, practically speaking, always horizontal; not only by reason of the basic origin of the disease, but largely also owing to the lowering of the diaphragm on the sound side, with extension of the cardiac beat into the epigastric notch.

Attention to these general guides may often prove of greater value than a close search for points of difference in the auscultatory and percussive sounds.

Percussion in advanced cases may yield different results in the same chest at brief intervals of time, according to the amount of retained secretion; and this variability is perhaps the most distinctive feature obtainable by the method. If in a chest otherwise resonant patches of dulness be found scattered in the middle and lower third, and particularly over the back, and if some of them yield a cracked-pot sound, a strong suspicion of bronchiectasis will arise. The high-pitched, the tympanitic, the amphoric, the splashing, and other varieties of percussion note which have been described cannot be expected in every instance. Much emphysema may almost preclude a diagnosis by percussion alone; although with a previous knowledge of the existence of sacculations their site could in most cases be made out by an expert percussor. The strong element of dulness in the fibroid variety of the disease, coupled with the boxy note obtained over the cavities when empty, is a much more definite guide; although the diagnosis from a basic cavity of tuberculous origin would still have to be made.

Auscultation, although not always capable of establishing a diagnosis between slight bronchiectasis and bronchial catarrh, seldom fails to identify advanced dilatation, from a joint observation of the respiratory sounds and of the râles.

As regards the respiratory sounds, the peculiarity of the emphysematous variety of bronchiectasis is the intimate blending of the tubular with the vesicular breath-sounds; the fibrotic variety is distinguished by the local absence of the latter.

The râles occurring in small dilatations, and in those which are mainly cylindrical, do not differ from ordinary catarrhal râles of medium and of large size. A distinctive character belongs to those produced in the sacculations. The sound, which is best described as "croaking," is partly due to the valvular action of the viscid and confluent secretion, and partly to the free communication and continuity subsisting between the sacculations and the corresponding bronchus. The undiminished length of the latter, and the branches which open into it above the terminal sac, are probably additional factors. An explanation of the mode of production of this sound is suggested above in connection with Fig. 5. The croaking sound is most distinctly produced in sacculations surrounded with more or less spongy tissue. In the fibrotic variety the solid medium through which it is conducted to the ear imparts to it a more metallic character.

It is unnecessary to dwell upon the common catarrhal sounds, the sibili and the rhonchi, which may spread over the lung as a result of general bronchitis. They may complicate the diagnosis by veiling to a certain extent the diagnostic sounds which have been described, although they seldom mask them entirely.

The voice sounds sometimes supply definite data. Bronchophony and ægo-bronchophony are yielded by the extensive and multiple sacculations of a partly cirrhosed lung, and sometimes by those not surrounded with fibrous tissue, if sufficiently large and superficial. Hollowness of the voice sound would, however, disappear if the cavity were to fill completely. The vocal fremitus varies considerably in different cases, the pleura being unaltered in some, in others greatly thickened.

Diagnosis.—The diseases most likely to be mistaken for bronchiectasis are the various forms of bronchitis and phthisis. Less commonly the difficulty may be to distinguish it from emphysema, pulmonary gangrene, and cancer.

When originating in a *general bronchitis*, dilatation, in its earlier stages, can only be inferred. Subsequently fœtor of the sputum necessitates a diagnosis from fetid bronchitis or bronchorrhœa; and, apart from any previous knowledge of the case, the distinction may be extremely difficult if a general catarrh should coexist. In the absence of the latter, dilatation would be known by the localisation of the large râles in the situations which present some alteration of the percussion note; and the same observation would also be a help in the more complicated condition. Again, the mode of the expectoration, even more than the nature of it, might throw light on the case; although in fetid bronchorrhœa the expulsion of the bronchial contents is often sudden and paroxysmal.

Pulmonary gangrene, occurring in aged or broken-down subjects and preceded by a history of chronic bronchial catarrh, would suggest bronchiectasis culminating in ulceration. Most commonly the onset of pulmonary gangrene is sudden and marked by extreme prostration; that of gangrenous ulceration of a bronchiectasis is gradual. As pulmonary tissue is expectorated in both cases, our guides must be the clinical data and the clinical history. But commonly in bronchiectasis a gangrenous odour occurs apart from any tissue necrosis; and a fruitless search for elastic fibre would strengthen any direct evidence of bronchiectasis otherwise obtained, and any negative evidence as to the existence of broncho-pneumonic or tuberculous processes such as lead to gangrene.

The intra-bronchial ulceration of an *empyema* may closely simulate bronchiectasis. Its presence will be sufficiently indicated by the history of an absence or insignificant amount of expectoration prior to the bursting; and of the considerable relief given by the latter to the cough, dyspnœa, pain, and thoracic deformity. The expectoration of an empyema is usually distinguishable at first sight, by its freedom from mucus, from that of bronchial dilatation. According to Biermer, it contains crystals of cholesterin and of hæmatoidin. In any special case a physical examination of the chest would probably remove any lingering doubt.

Prior to the discovery of Koch's bacillus the diagnosis from *phthisis* had to be made almost exclusively from physical signs, and was often very difficult for persons unfamiliar with the physiognomy of bronchiectasis. A microscopical examination of the sputum now decides the question. Nevertheless, the other elements of diagnosis—(*a*) the clinical history, (*b*) the general clinical state and aspect, and (*c*) the physical signs—are too important to be neglected.

(*a*) In most cases phthisis can be traced back to characteristic beginnings, the constitutional effects of the invasion being out of proportion to the pulmonary symptoms existing at that time. This is not the history of bronchiectasis, which begins with a definite bronchial affection, or with a pneumonia or a pleurisy; the worse constitutional symptoms being relegated to the late stages. Again, when the patient's affection begins with a profuse hæmoptysis the probability of its tuberculous nature is great.

Moreover, the duration and the progress of the two diseases are strikingly different. Cough and expectoration of many years' standing, in a subject not markedly marasmic, would not be features of the common phthisis; though we should not forget that unilateral phthisis may, and often does, run an exceedingly protracted and mild course. In such a case the signs would be unmistakable and strictly apical, and therefore unlike those of bronchiectasis which, when single, hardly ever implicates the pulmonary summit.

(*b*) Between ordinary pulmonary tuberculosis and ordinary bronchiectasis a very marked contrast in the general clinical appearances is at once perceptible. In the ultimate stage of pulmonary consumption there is no difficulty in the diagnosis; the patient carries it written large in every feature. At a rather earlier period in the complaint, when doubt might be possible, the same peculiarities are apparent, although not yet so manifest as to strike the superficial observer. They are briefly these—wasting of the subcutaneous fat in general, and in particular of the fat of the orbit and of the cheek; wasting of the muscles; visible loss of energy; pronounced anæmia, in the strict sense of the word, namely, reduction in the total amount of the blood, the patient being bloodless and withered. These are not features of bronchial dilatation, uncomplicated with tubercle, at a like interval after the beginning of the affection: emaciation usually exists, but it is not extreme; there may be slight anæmia also, but it does not confer the characteristic wan look of phthisis. The hollow orbit, with undue exposure of the sclerotic, the sunken cheek with projecting malar eminence, and the thin, drawn lip are all conspicuously absent. Instead of these bronchiectasis often presents outward peculiarities of its own; a certain fulness of the eye, of the lip, and of the features, and a slight duskiness of the complexion suggestive of congestion rather than of anæmia: and the veins, the jugulars in particular, are commonly conspicuous, if not turgid. On analysis these peculiarities will be found correlated with the state of fulness of the right side of the heart, which in advanced phthisis is never surcharged, in spite of the great obstacle to the pulmonary circulation. In short, the bulk of the blood is not reduced in proportion to the pulmonary destruction, as is the case in phthisis. For the same reason also the depressed and devitalised aspect peculiar to phthisis is not noticed in this disease.

Another striking peculiarity is the unusually bulbous expansion of the finger-tips, associated with a very marked incurvation of the nails. In phthisis the nails are aduncate, but the finger-ends are seldom much clubbed; nay, the pulp of the finger is often wasted.

Prognosis.—The spontaneous cure of acute bronchiectasis, such as it occurs in the growing lung of infants, cannot be expected at a later age; and a restoration of the damaged lung is impossible. In rare instances, where the dilatation is single, and where it is no longer the seat of catarrh, as in the exceptional case of the cicatricial closure of its bronchus higher up, the disease may become obsolete. Lebert quotes a case of Bamberger's, in which the formation of an external fistula eventuated in a cure; and a similar result might be hoped for from the surgical treatment of a solitary dilatation. In the great majority of chronic cases, so long as the original conditions persist, the disease, if left to itself, is inevitably progressive; and therefore less likely as time goes on to be permanently relieved. The most favourable achievement to be gained by treatment is often no more than a relative quiescence of the trouble; or a reduction in the rate of a progressive destruction of the lung.

As regards duration of life, the great diversity in the kind, degree, and multiplicity of the lesions, and of their bronchial, pulmonary, and pleural complications, must establish a wide difference between the chances. Of this some idea is given by the figures obtained by Lebert in a series of fifty-two cases.

The period of survival was :—

Of one year	in 21·1 per cent
Of one to two years	„ 7·7 „
Of three to five years	„ 30·7 „
Of six to ten years	„ 15·5 „
Of upwards of ten years	„ 25·0 „

Apart from all other difficulties, an exact determination of the extent and number of the lesions is so unlikely to be attained by physical examination, that the physician's forecast in the individual case must be based on very broad considerations: such as the age, temperament, antecedents, energy, nutrition, and general circumstances of the patient; the unilateral or bilateral character, and the cirrhotic, emphysematous, or stenotic type of the affection; the presence or absence of heart, kidney, or liver disease; the present and the previous state of the expectoration, and the effects of treatment on the catarrh.

Often enough, when all has been taken into account, great uncertainty must still surround the prognosis, and it will be wise not to venture upon too precise a statement of the probabilities. In the future much more may be expected from an improved diagnosis, and from the earlier adoption of improved preventive, palliative, and curative measures, than from surgical interference, which is not likely to prove more successful than in the past.

The worst prognosis will probably always belong to the bilateral cases and to the unilateral cirrhotic variety, especially when associated with some defect of the other lung or pleura. Hæmorrhage is occasionally a fatal complication; it is apt to be profuse in cases of valvular disease or of secondary cardiac dilatation. The occurrence of perforation and pyopneumothorax, or of ulceration with the attendant dangers of gangrene, of putrid bronchitis, of pyæmia, and of septicæmia, would justify a grave prognosis. Mere fœtor of the expectoration is not in itself an alarming sign.

In those most favoured cases which remain free from all serious complications life may not be greatly shortened.

Treatment.—The acute bronchial dilatation of early childhood, depending upon temporary impairment of the expansion of lobules, and of the pulmonary and bronchial elasticity, is capable of spontaneous recovery. The general principles on which such cases should be conducted are too well known to need comment.

Inveterate bronchiectasis, though not, strictly speaking, curable, is often susceptible of considerable amelioration. The extent to which positive results may be hoped for must largely depend upon the extent of the bronchial lesions, and especially upon the condition of the surrounding tissue; the most unpromising cases being those in which ulceration or considerable fibrosis has already taken place.

In addition to the therapeutic measures specially intended for the pulmonary condition, we shall consider those meant for the relief of complications and for the improvement of the constitutional state.

The constitutional treatment, an essential adjunct of the pulmonary treatment, need not detain us long, since its climatic and hygienic aspects are included in the account to be given of the latter. It cannot be regarded as curative, nor even as being aimed at the cause of the affection; but it undoubtedly promotes the patient's chances and the results to be obtained from symptomatic treatment. The only instances in which it might claim to be in any sense specific are those in which the disease has been traced to syphilis, and in which mercury, a drug possessing also general advantages as an antiseptic, should have a trial. Iron, quinine, and cod-liver oil perseveringly administered, with intervals of rest and interludes of hepatic treatment, are still, so far as we know, the best means to the end of strengthening both fibre and function. Syrup of the iodide of iron in liberal doses, or the hypophosphites of calcium, of sodium, and of iron also freely administered, are remedies specially adapted to counteract the exhausting effect of catarrh on the serous and glandular elements. A liberal, varied and nutritious diet, and a moderate allowance of burgundy or of port wine are indicated. Much general tonic effect may also be obtained by systematic treatment of the skin and by salt-water baths—subjects to be discussed presently. Neither should we lose sight, in cases showing a tendency to venous stasis and to cardiac dilatation, of the great value of derivative, alterative, and mildly hepatic treatment. Much might be effected in early stages by hygienic and medicinal measures of this kind; but too often the opportunity of recommending them is not afforded until it is almost too late for their successful employment.

The treatment of complications.—As in other chronic affections, medical advice may at first be called in for the treatment of aggravated symptoms, of complications, and of emergencies. Among the latter, hæmorrhage—fortunately rare in its worst form, that of ulcerative perforation of an arterial branch—calls for immediate action, and must be treated on the usual principle of reduction of blood-pressure, by subcutaneous injections of morphia, by calomel by the mouth, and by an enema of glycerine (not of a large bulk of fluid); all of which should be administered as soon as possible.

The febrile exacerbations of the bronchial catarrh, the complications of pneumonia and of pleurisy, the severe symptoms attendant upon absorption of septic material, and the occurrence of ulceration, with threatenings of gangrene, will need measures adapted to each event. In all of them a supporting plan of treatment will be necessary, and, in those last mentioned, stimulants, both medicinal and alcoholic, must be freely administered.

The special treatment of the respiratory organs should be guided by the following indications: (i.) the emptying of the cavities; (ii.) the relief of the fœtor; (iii.) the reduction of the catarrh; (iv.) the protection of the membrane from further irritation; (v.) the diminution of the size of the dilatations, and (vi.) the improvement of the respiratory function in general. Until recently these indications have been very imperfectly fulfilled. The methods employed have acted as palliatives, but their inability to check the progress of the worst cases has been one of the reproaches of medicine, and has led within recent years to a desperate resort to surgical measures, the hopelessness of which has now been made apparent; and indeed was almost foretold in the anatomical peculiarities of the affection.

As regards the emptying of the dilated tubes, sufferers often discover at an early stage the value of posture as a mechanical aid to the bronchial outflow. With the majority, lying down or turning to one side or to the other will bring on more or less cough and expectoration; but in others, when the dilatations are situated at the back, it is the change to the sitting posture which induces the paroxysm of cough. In this disease, even more than in phthisis, lowering the head, either over the edge of the bed or whilst standing, will allow the accumulated secretion to gravitate out of the sacculations and into the receiver. Some patients are in the habit of practising this method of relief. Its regular employment should be suggested whenever no contra-indications exist. In the case of multiple bronchiectasis systematic treatment should also include, unless there be good reason to the contrary, the yet more effectual resort to an emetic; and it is well to administer, for two days prior to this, repeated doses of an expectorant mixture containing ipecacuanha, small doses of vinum antimoniale, and iodide of potassium,—a mixture to be subsequently continued until a second emetic shall have been taken at an interval of a few days. The object is to wash out the stale secretion by a more abundant flow of watery mucus. Much will have been gained if this result can be secured.

For the relief of the fœtor two methods have hitherto been adopted alternately or combined: (*a*) the inhalation, and (*b*) the internal administration of deodorising and antiseptic agents.

(*a*) Inhalations as a rule fail to influence the bulk of the accumulations, though they may reach the uppermost layers. A noteworthy exception must be made in favour of those inhalations which set up cough and copious expectoration.

Theoretically, oxygen was expected to fulfil a double purpose, as an aid to respiration and as a disinfectant; but it has really proved of little service, partly perhaps because of its tendency to diminish rather than to increase the activity of the respiratory movements.

Some relief may be obtained from the inhalation, from a jug, of vapour impregnated with thymol, eucalyptol, wool fir oil, or other antiseptic.

Inhalation may also be practised with the dry inhaler, through which air is drawn over a sponge or a quantity of cotton wool steeped in the solution to be used. Since only those constituents are inhaled which are volatile at the ordinary temperatures, substances such as carbolic acid, creasote, tar, terebene, and others can be used fairly concentrated. Iodine can also be used with proper precautions.

Lastly, inhalation may be conducted on the principle of the spray. Steam sprays, at one time much in use, have their drawbacks, but in some respects are convenient: they may be made the vehicle of a great variety of medication. The complication of steam is avoided in the mechanical spray-producers which "atomise" the solutions to be inhaled, by forcing them through the minute orifice of the outlet with a jet of compressed air worked by an india-rubber hand-ball. In this case the solutions are not diluted by steam, and must be prescribed of an appropriate strength. The dripping and dampness inseparable from the steam are avoided; and the nozzle of the instrument can be introduced into the nose or mouth, thus almost ensuring actual inhalation of a large proportion of the remedies. The finest subdivision is obtained—as in Oppenheimer's "nebuliser"—by combining strong pressure with smallness of orifice. The latter condition unfortunately limits the supply of the medicated atmosphere.

(*b*) The internal administration of creasote, tar, terebene, the essential oils, the oleo-resins, and the balsams has long been in use. Only of late years, however, have the improvements in pharmaceutical detail enabled efficient doses of the more powerful of these agents to be taken with comfort. Copaiba, tar, and especially thymol, eucalyptol, guaiacol, and creasote, can be administered in the shape of capsules at frequent intervals throughout the day; and, by the persistent action kept up on the respiratory mucous membrane, may be of great benefit. Fifteen centigramme capsules of myrtol, taken every two hours throughout the day, are well spoken of in Germany; and are worthy of trial in cases in which none of the measures about to be described can be carried out.

The fault of most of these methods is their inadequacy; they do not deal with the evil at its chief seat in the depths of the lung. A new era in the prognosis of bronchiectasis has happily been opened up by the more thorough methods associated with the names of Dr. Vivian Poore, Sir T. Grainger Stewart, and Dr. Arnold Chaplin; these methods consist respectively in the internal administration of garlic, in the intralaryngeal injection of disinfecting solutions, and in the systematic inhalation of the vapour of coal-tar creasote.

(1) Dr. Poore's method is based upon the penetrating properties of some of the volatile constituents of garlic, and upon their stimulating and antiseptic as well as odoriferous virtues. Garlic probably acts as a general tonic as well as a local stimulant. Its local effect is produced at the surface of the mucous membrane by exhalation; but the fact that the smell of garlic is also given off by the skin suggests that the constitutional influence of the drug may be widespread and important.

The favourable results reported by Dr. Poore in his work on *Nervous Affections of the Hand and other Studies* were obtained from the continued administration of sufficient garlic to render the odour permanent in the breath. In the cases to which he refers the original fœtor of the expectoration was replaced by a pungent smell reminding one of that of syringa. The discharge was greatly diminished; and a remarkable improvement took place in the health, in the strength, and in the weight of the patients. The treatment is generally well borne, and, if the remedy be taken with meals, patients submit to it without much inconvenience. A clove of garlic is chopped up and mixed with the beef-tea, or preferably enclosed in gelatine capsules. I have administered as much as eight capsules daily, each containing thirty grains of chopped garlic. An extract might also be used. Dr. Poore suggests that sulphide of allyl, which is contained in the essential oil of garlic, is probably the remedial agent. The oil of allyl has an exceedingly penetrating smell. It should be taken immediately after meals. I have prescribed it in three-minim capsules three times a day; but this dose is too large, and soon disagrees. Capsules containing half a minim of the oil will be found more convenient. The remarkable results obtained by this method are not limited to cases of bronchiectasis, but have also been obtained in phthisis. The chief theoretical objection to the treatment by garlic is that, whilst it provides for the disinfection, it does not ensure the complete clearance of the dilated bronchi, nor directly assist their contraction.

(2) Intratracheal injection, suggested years ago, and experimentally tried in animals, was first performed with the hypodermic syringe,—a valuable method in some cases, and was described by Dr. Sehrwald.

The idea was applied in a practical form to the treatment of pulmonary affections by Rosenberg, Colin Campbell, Jamieson, Downie, Byrom Bramwell, and by Sir T. Grainger Stewart, who treated with great success by the intralaryngeal method an inveterate case of fetid bronchiectasis in which all other measures had failed. The treatment consisted in injecting twice daily into the trachea (through the glottis) one drachm of a solution of 10 parts menthol and 2 parts guaiacol in 88 parts of olive oil. The injections were continued for a considerable time with benefit.

This method, which has now been fairly tried and seems likely to lead to important results, is the only one which aims at disinfecting the secretions in the lung by fluid admixture with the disinfectant, and at treating the mucous membrane locally by soluble applications. What proportion of the injection may reach the dilatations will depend upon circumstances not easily controlled, but chiefly upon the posture adopted by the patient at the time of the operation and afterwards. At any rate, the effect on the bronchial mucous membrane must be widespread and decided, and, with a systematic use of the treatment, would finally extend to that of the diseased mucous membrane also. The possibilities opened up by this therapeutic innovation are obviously great, and its applicability is not restricted to the disease under discussion, nor to the stated formula. Chronic bronchial catarrh, fetid or putrid bronchitis, and bronchorrhœa, especially of the purulent variety, are suited for its adoption.

Although in common with those who have tried this method I have wondered at the facility with which the pulmonary lymphatics dispose of the injected solution, we are left too much in the dark as to the destination of the latter. It is much if, by carefully directing the nozzle of the syringe and adjusting the patient's posture, we can ensure the treatment of one lung rather than of the other; but we are unable to control the injected fluid in its course down the tubes. In the majority of cases nothing but good happens. In tuberculous cases, however, there may be some misgiving as to the possible dissemination of the infection from the upper into the lower lobes. From personal observation I regard the use of the method in cases of phthisis with grave suspicion; and it has been my regret to witness, after its repeated use, the appearance of persistent râles, of bronchial engorgement, and of catarrhal pneumonia at both bases in a case which ended fatally.

(3) No objections of this sort can be urged against Dr. Arnold Chaplin's creasote method, which both theoretically and in its results is the only one hitherto claiming to be strictly rational and thoroughly adequate. Its principle is to obtain an amount of coughing sufficient to squeeze out every remnant of the noxious secretion, and to keep up local disinfection by inhalation for a sufficient time, and in sufficient strength, to enable the mucous membrane and the lung itself to be completely purified. These indications once fulfilled, nature will do the rest. Living in an atmosphere of the disinfectant would carry out an important part of the treatment; and Dr. Chaplin originally noted the tradition, which exists among workmen constantly employed in an atmosphere of creasote, that the fumes "clear the chest of phlegm," and confer an immunity from "asthma" and consumption. But in bronchiectasis the object is to bring about a complete expectoration of the bronchial contents; and with this view the creasote atmosphere has to be made almost intolerably strong, so that it can be inhaled for short periods only. This concentration of the vapour is the irksome side of the treatment; but any objections connected with the hardships of the method will, after a brief trial, be laid aside when the patients have experienced the remarkable relief afforded by its use. In addition to the intense cough, which has the advantage of leading to inhalations of the disinfecting agent proportionately deep, the discomforts are chiefly the irritating action upon the other mucous surfaces and the eyes, the strong smell which clings to the hair and clothing, and the diffusion of the smell into the surrounding space. So pervading is the odour that it may be complained of even within neighbouring houses, and it is desirable to provide an entirely separate inhalation chamber at some distance from the doors and windows of other buildings. The remaining difficulties are met by loosely plugging the nostrils with cotton wool, by wearing over the eyes watch-glasses framed in bandage or sticking-plaster, and by covering the garments and the head with oiled silk or mackintosh.

The inhalation chamber should be of small size, 6 or 7 feet wide by 8 feet high, and should be made as air-tight as possible, with cotton wool or tow, in order to obtain a dense creasote atmosphere. In vaporising the creasote proper care must be taken to prevent a conflagration. A fair-sized metallic evaporating dish is the best, and into this it is convenient to place some dry sand. Some more stable support than the common tripod should be used, and gas flames must be avoided.

At the first sittings the patient may with advantage enter the room before the spirit lamp is lighted under the dish containing the sand and creasote; but subsequently time may be saved by vaporising beforehand some of the creasote. The duration of the exposure is gradually increased from a quarter of an hour to an hour or more. The residual phlegm dislodged by the searching cough is exceedingly offensive; but the fœtor is partly covered by the strong creasote odour. The treatment, unless contra-indicated, is to be continued daily until little is coughed up in the chamber, and until no expectoration is brought up spontaneously the next morning. In an average case this will imply a treatment of from four to six weeks.

In itself the adventure is a valuable respiratory exercise. Whilst the cavities are being cleared and disinfected collateral expansion of the lung is induced by the cough, and the gradual contraction of the sacculations is promoted. A remarkable improvement takes place in the general health and strength, as well as in the respiratory capacity. In the seven cases originally reported by Dr. Chaplin excellent results were obtained. Notes of equally successful cases have been kindly communicated to me by Dr. Devereux of Tewkesbury. A full account of one of his cases has been published by Dr. Brian Dobell. Through the kindness of Dr. Dobell and of the Editor of the *British Medical Journal* the temperature chart of this case is reproduced on the following page as a striking illustration of the reduction of temperature which is obtained in pyrexial cases.

A case of inveterate bronchiectasis under my own care is for the present cured. In another the relief seemed to be complete, but was followed by a relapse which did not yield thoroughly to a second course. Reduction of temperature and of the expectoration and general improvement were also observed in a third patient with fibroid disease of the lung and profuse offensive expectoration; but the treatment has been temporarily interrupted, whilst these pages are being written, because of an intercurrent aggravation of the catarrh, due perhaps to irritation by the fumes.

The freedom from risk and the brilliant results hitherto secured in most of the cases reported leave us no choice: so long as we have no better method, every sufferer should have the benefit of a trial of the inhalation method. A combination with it of the administration of garlic, whilst adding a fresh therapeutic infliction to a trying treatment, would probably curtail the duration of the latter. Lastly, for cases not yielding sufficiently good results the intralaryngeal injection method would still be available.

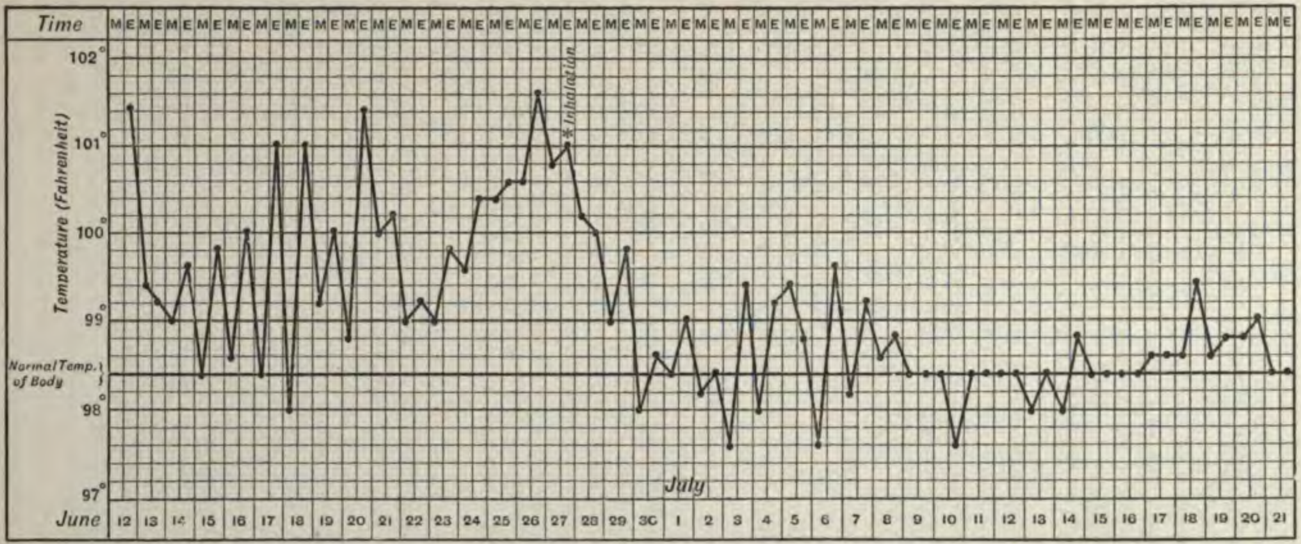

CHART 1.—Reproduced from Dr. Brian Dobell's report of Dr. Devereux's case of bronchiectasis, showing the influence of the creasote inhalation treatment upon the pyrexia.

The prospect of a permanent cure will be greater the earlier the creasote treatment can be applied. Some of the inveterate cases which have long resisted all other remedies may fail to end in a complete recovery, and may need repetitions of the course. But their number will decrease as the method comes into more general use. Indeed, it is not improbable that in the future, when cases are treated sufficiently early, bronchiectasis may cease to be regarded as an incurable disease.

Surgical treatment.—An attempt was made some years ago to treat the lesions by injecting weak solutions of carbolic acid, of iodine and of other antiseptics through the chest wall into the surrounding pulmonary tissue. No good results were obtained by this method, which has since then been almost forgotten.

Treatment by incision and drainage was proposed and attempted as a last resort a few years prior to the recent advances. Although this may now be regarded as a closed chapter in the history of pulmonary therapeutics, it calls for a few retrospective remarks. The few cases of operative interference which have been reported in this country and elsewhere are not encouraging. Hofmockel, who gives a review of eighty cases of operation for abscess, for gangrene, or for bronchiectasis, finds that the worst results were obtained in the cases of bronchiectasis.

A disastrous experience has shown that success can be looked for only where a single dilatation exists. These are, however, the cases in which the symptoms are least urgent as well as least refractory to the ordinary measures. Where help is most needed—in the instances of multiple sacculations—surgery is doomed to failure. To attempt multiple incisions is to multiply the risks of septic infection of the pleura; and to open only one of the sacculations is not only to leave a great part of the disease unrelieved, but to place its remaining foci in a worse position than before, by weakening the expiratory mechanism of cough owing to the direct leakage of air, and to the unavoidable interference with the freedom of the thoracic movements.

The *mechanical hygiene of respiration* and the *climatic treatment* may be considered under one heading. They are both necessary adjuncts to any method of treatment, although in the future their relative importance will probably be less than it has been heretofore.

For the control of the catarrh and for the protection of the mucous membrane from further irritation we had until recently looked with greater confidence to the effect of climate than to medicine. The great indication was to strengthen the clogged and sodden mucous membrane by constant contact with the purest air, whilst invigorating the system by prolonged residence in a warm and equable climate, where patients might live in the open. The dry and stimulating climates to be found on the table-lands of South Africa, in South California, on some of the slopes of the Andes, or at high elevations in islands, as in the West Indies, or even in the Mediterranean, as at Ischia or Capri, are specially indicated; and along the shores of the Mediterranean there is a large selection of suitable sites. For a fuller discussion of this part of the subject the reader is referred to the chapter on "Climate and Disease" in the first volume of this work.

Patients who at a sufficiently early date adopt and adhere to this thorough treatment by climate might hope for a permanent arrest of their catarrh, and, thanks to the increasing pulmonary expansion due to open-air life, might ultimately secure a degree of improvement almost equivalent to a cure. For this happy result a life-long treatment is now less indispensable, nor need we expatriate our patients. The climatic treatment is henceforth, as in the case of other diseases, an after-cure. A suitable climate for the winter retains its importance; but its selection is no longer limited as formerly when the consequences of any incidental catarrh were much less within our control. We might, for instance, without serious risk, in the case of some convalescents not advanced in years, recommend the dry, cold atmosphere of the Alpine winter and the outdoor life and physical exercise, which are not the least of the advantages of the Alpine cure; whilst for those unable to travel our home resorts and seaside places afford eligible climates, among which Thanet, Folkestone, Eastbourne, and Brighton deserve special mention.

Warm sea-water baths may be of considerable value. For some patients a stronger effect might be sought from the artificial Nauheim salt-water baths. In any case the temperature and the duration of the bath must be adapted to the individual. An important part of the balnear treatment is the tepid, and ultimately the cool or cold affusion terminating the bath, followed by strong friction of the surface.

Among the *medicinal springs* the sulphurous thermal waters enjoy a deserved reputation in the treatment of this affection. Harrogate, Moffat, Challes, Aix-les-Bains, Eaux Bonnes, Eaux Chaudes, Cauterets, Bagnères-de-Luchon, and a variety of other spas might be visited with profit; but for patients unable to leave home a substitute may be found in tonic baths combined with the internal administration, for recurring periods, of some preparation of sulphur.

At most of the foreign health stations and at some of our own various *hygienic measures* are recommended in addition to the use of baths or waters.

Among them special value attaches to the following:—

(*a*) The inhalation of an oxygenated and terebinthinated atmosphere; (*b*) systematic exercise, at first passive only, of the thoracic muscles and of the abdominal muscles, including the use of dumb-bells or clubs, and a variety of postural exercises; (*c*) systematic respiratory gymnastics, such as deep inspirations followed by deep expirations in various attitudes, reading aloud or singing; (*d*) general massage and passive resistance movements followed by brisk rubbing. An improved circulation through the skin and a general bracing of its nerves are special objects of this form of treatment; another is the tonic effect on the right heart and pulmonary circulation, and the help which the mucous membrane may derive by sympathy from a healthier cutaneous surface, and from its improved reaction to atmospheric influences.

The importance of these systematic methods lies in the regularity with which they can be enforced; but the benefit they can confer might equally well be secured by a perpetual out-door life in a really suitable climate, and by progressive exercise gradually pushed to the extent of slight breathlessness.

The contraction of the sacculations and the general improvement of the respiratory function, which are the final aims of our treatment, are directly promoted by all the measures which have been detailed; and in none of the ordinary cases, nor even in fibrotic cases if one lung be perfectly sound, need we despair of their partial attainment.

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PNEUMONIA

Definition — Nomenclature — Historical sketch — Clinical course — Symptoms and physical signs — Diagnosis — Complications and sequels—Secondary Pneumonia and other Clinical Varieties—Morbid Anatomy — Histology — Bacteriology — Pathology — Etiology—Prognosis—Treatment—Statistics of four hundred and thirty-five cases—References—Catarrhal Pneumonia—Chronic Pneumonia.

SYNONYMS. — *Peripneumony*, *περιπνευμονία*, Hippocrates and later Greek writers.[1] *Pneumonia*, *πνευμονία* (Attic form *πλευμονία*, used by Plutarch). *Peripneumonia vera* (Sydenham), as distinguished from *Peripneumonia notha*, "obstruction of the lungs by a heavy, viscid pituitous matter," that is, in modern nomenclature, bronchitis. *Pneumonic fever* (Huxham). *Pleuro-pneumonia*; *Acute* or *Sthenic pneumonia*; *Lobar pneumonia*; *Croupous* or *Fibrinous pneumonia*; *Pulmonary fever.*

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[1] "Vehemens et acutus morbus quem *περιπνευμονικόν* Græci vocant."—Celsus, lib. iv. cap. vii.

Definition.—A febrile disease, running a short course, with a special form of acute inflammation of one or both lungs.

The disease which is now understood by the name Pneumonia, when stated without qualification, has been a common acute disorder in all historical times, in all climates, and at all periods of life. It is one of the most striking and characteristic of maladies in its symptoms and physical signs, and not less so in its morbid anatomy. Other forms of acute inflammation of the lung bear the same name; but they bear it with a difference: they are, or should be, always distinguished as broncho-pneumonia, lobular, or catarrhal, hypostatic, tuberculous, or pyæmic pneumonia. These all differ pathologically and clinically from the disease now under consideration; and no less different is a chronic interstitial inflammation of the lungs—also known as iron-gray or slaty induration—which is sometimes called chronic pneumonia. It would be well if the historical name pneumonia could be restricted to the acute disease with lobar hepatisation; and the other inflammatory affections of the lung be named pulmonary catarrh, pulmonary abscesses, and cirrhosis of the lung.

History.—Before morbid anatomy was studied, or physical diagnosis invented, acute inflammations of the chest, whether affecting the parietal pleura alone or the lung with its pleural covering, were described under the name peri-pneumonia; that of "pleurisy" being applied to the sharp characteristic pain in the side which accompanies both diseases. Charlemagne is said to have died of a "fever, with a pain in the side which the Greeks call pleurisy." We now recognise the pain as due to inflammation of the pleural membrane, and the name of the symptom is applied to the anatomical change which it accompanies; pleurisy always accompanies acute lobar pneumonia, although it is often present independently; and the name pleuro-pneumonia is therefore superfluous.

The characteristic clinical features of pneumonia were identified with solidification of the lungs by Morgagni. Baillie described them as sometimes converted into a solid mass very much resembling liver (hepatisation). But Laennec, Cruveilhier, and Rokitansky completely described the anatomy of the disease. The diagnosis of pneumonia by auscultation was one of the most important results of Laennec's great discovery. The chief steps since made in advance have been the proof by Addison that the exudation of pneumonia is not into the "interstices" of the lung, but into the air-vesicles themselves; the distinction between fibrinous or lobar and catarrhal or lobular pneumonia, which is due to Rokitansky; and the discovery of a specific pathogenetic microbe, which has been the work of numerous observers.

Clinical features of the disease.—*Onset and early symptoms.*—The symptom which commonly marks the onset of pneumonia is a feeling of chilliness, a fit of shivering. When this initial rigor has passed off, the patient often feels prostrate, with headache but without the severe lumbar pains which mark the onset of some acute diseases. He becomes thirsty, the skin is hot and dry, and the pulse is quickened; the temperature rises rapidly from the beginning, and by the evening of the first day often reaches 103°, or in children 104°.

When twelve or more hours have passed from the onset of the disease the patient's aspect is characteristic; the face is flushed, the eyes bright and watchful, the expression that of constraint and apprehension. He usually lies on his back without marked orthopnœa. Breath is short, frequent, and shallow, deep and efficient respiration being hampered by a sharp pain in the side; the pulse is quickened, full, and of increased tension, but varies less from the normal than the temperature, and still less than the breathing. The skin is dry and pungent. Except scarlatina, and ague in its second stage, there is scarcely any disease which gives such a sensation of burning heat to the hand of the observer, a character probably due to the absence of perspiration.

Not infrequently an eruption is seen upon the face, which consists of a group of small, clear vesicles on a reddened patch. This *herpes labialis* is most commonly seen on the upper lip in the neighbourhood of one or the other nostril; but it may be seen on the lower lip at the angle of the mouth, or upon any part of the cheek, chin, or jaw; hence it is sometimes called herpes facialis. Although frequent enough to form a characteristic feature of pneumonia, it is probably not present in more than two-thirds of the acute idiopathic cases we are now describing; and it is the exception instead of the rule when pneumonia is secondary to another disease. Moreover, exactly the same eruption may occur with slight pulmonary catarrh, or bronchitis without bronchopneumonia, or with a mere cold in the head. Indeed some persons have an attack of herpes labialis with almost every accidental catarrh. It is occasionally seen in other febrile attacks also; or it may even follow a rigor without subsequent fever. The little vesicles cause no irritation or pain; they become purulent and dry up, leaving their dark crusts, which drop off and leave no trace. The eruption lasts from ten days to a fortnight.

As in other febrile disorders, there is complete loss of appetite, with constipation. By the second or third day the tongue is thickly plastered with white fur, particularly if the patient is kept on a diet of milk. It closely resembles the appearance of the tongue at the beginning of acute rheumatism and of scarlatina.

Physical examination of the chest during the first few hours will often determine the nature of the case by the presence of a pleuritic rub, or a small crepitant râle; and towards the end of the first twenty-four hours, or on the second day, consolidation of a part of the lungs will be recognised by its physical signs. These will be conveniently considered together after the account of the external symptoms has been completed; but it must be stated here that the evidence of physical changes in the lungs, derived from auscultation, is occasionally absent on the first, the second, and even the third day. In these cases there is no reason to suppose that the local change is present without its appropriate signs;

nor are we aware of any such case in which the opportunity occurred of ascertaining the state of the lung; in the absence of such direct observation, we are justified in believing that the pneumonic fever with all its characteristic symptoms may precede the pneumonic hepatisation not only by hours but by days.

Clinical course.—The *temperature*, after an abrupt and rapid rise on the first day of the disease, continues at the degree then reached, or rises somewhat higher; the mean range being from 103° to 104°. In severe cases it may reach 105° and upwards; while in slight cases, particularly in children, it is often a degree lower. The same is true, even of severe cases, in elderly people. The moderation of the pyrexia, not uncommon in children, depends on the rule that the mildest cases of pneumonia, as of enteric fever, occur in children; the lower temperature in elderly patients on the still more general rule that the same degree of febrile disturbance, as measured by other symptoms and by local changes, will produce a higher temperature in a child or young adult than in an aged patient. The morning remission and evening rise, which are rarely quite lost in any case of pyrexia, are present in pneumonia; but they are less marked than in enteric fever, and still less than in suppurative fever. Hyperpyrexia is not an uncommon event; some cases indeed, and these not always the most severe in their subsequent course, manifest a very high temperature on the first or second day. The *pulse* usually lags behind the rising temperature, and the *respiration* follows rather the extent of the local changes in the lungs than the course of the fever. (Appendix, p. 134.)

Sometimes at the very beginning of the attack, sometimes later on the first day or in the course of the second, the patient feels a sharp *pain* like a magnified stitch in the side. This pain, independently of auscultation, tells us which lung is affected; for although the pain is pleuritic, the pleurisy is part of the pneumonia. It is usually referred to the front of the axilla an inch or so outside the nipple; sometimes to the post-axillary line a little outside the angle of the scapula; occasionally it is felt in the mid-axilla, and still more rarely towards the base of the lung. A friction sound can usually be heard at the seat of pain; but inspection of a pneumonic lung after death shows that the pleural inflammation is more extensive than the point on the chest-wall to which the pain is referred. It is certainly rare to hear a rub under the clavicle, or above the scapula in cases of apical pneumonia; and the same remark applies to the pleurisy which so frequently accompanies tuberculous disease of the apex.

The pain felt on taking a deep breath makes the breathing shallow and hurried, without dyspnœa; and the cough is short, dry, and restrained by the patient.

The *urine*, from the first day onward, shows the characters of pyrexia in a marked degree. It is like that of rheumatic fever, of quinsy, scarlatina, or typhus; scanty, high-coloured and very acid. The lithates of soda and potash are deposited as it cools, partly from want of water to dissolve them, partly from a strongly acid reaction, and partly also from an increase in the output of uric acid. The daily excretion of urea is also larger than in health.

The inorganic salts excreted in the urine—the phosphates, sulphates, and chlorides, and particularly the chloride of sodium—are diminished as in other febrile disorders; but in a much greater degree. When nitrate of silver with excess of nitric acid is added to the urine of pneumonia, it is not uncommon to see an opalescence only in place of a thick opaque precipitate. This diminution of the saline constituents of the urine does not depend merely on the patient's diet, as experiment has proved, but is probably due to the abundant exudation of salts into the lungs. Hepatised lung is found to contain considerably more saline constituents than healthy lung, and during convalescence there is excess of salt in the urine.

Another feature of febrile urine in general is particularly frequent in pneumonia, namely, the presence of albumin. This has been observed by different authors in one-third, one-half, or two-thirds of the cases. It is probable that even the last estimate is below the truth, and that some amount of albumin is present in almost every case of primary acute pneumonia.

The sputum which the patient coughs up is characteristic. It is not abundant, not very frothy, and is unmixed with saliva or with pus. It consists of clear, tenacious mucus with a few air-bubbles, and is more or less deeply stained with blood. It hangs on the patient's lips and clings to the vessel in which it is received. The colour, when most characteristic, deserves its traditional name of "rusty"; it is a bright orange-brown, resembling the burnt sienna of the colour-box. When the sputum is abundant and thin it loses its bright chestnut colour, and has been aptly compared to the juice of dried prunes when stewed. Under other conditions the reddish tint is lost and is replaced by a greenish yellow; the sputum is then compared in colour to greengages. When the amount of blood is scanty, a bright lemon colour is no less characteristic than the more usual rusty sputum; when, on the other hand, it is excessive, the yellowish brown is replaced by a more decided red, and in some cases pure blood is spat up. This hæmoptysis is sometimes very free, and denotes, we may presume, unusually intense congestion or unusual fragility of the pulmonary vessels. Whatever its immediate cause, it does not appear to have any unfavourable import, and certainly it does not point to subsequent tuberculous disease. But the "prune-juice" and the "greengage" varieties of pneumonic sputum are justly held to be of graver significance.

On microscopic examination, beside transparent structureless mucus with a few small air-bubbles, the sputum of pneumonia is found to contain abundant blood-discs, a few leucocytes, and frequently minute casts of the smallest bronchioles. When treated by appropriate staining, the pneumococcus or diplococcus described by Fraenkel—an oval or rounded organism with a thick transparent capsule—is revealed; some of them are separate, some in groups of three or four; but almost every slide will show several pairs united two and two in characteristic fashion.

The rusty sputum has of all the symptoms of the disease the best right to be called distinctively pneumonic. Its peculiarity is due to the hæmorrhagic quality of the inflammatory exudation itself, which may be compared in this particular to that of acute glomerular nephritis. As in the smoky urine of this disease so in the rusty sputum the blood-discs have been poured out uniformly and continuously from the first, and are therefore more equally distributed than when hæmorrhage is added to inflammation as a subsequent event. Accordingly we do not find the characteristics of "rusty" sputum in the hæmoptysis of phthisis, of aneurysm, of purpura, or of laryngeal or tracheal ulceration. Nor is it, as a rule, to be seen in cases of cardiac disease with consecutive pulmonary hæmorrhage. In these cases the blood is more separate from the sputum. When, as sometimes happens, characteristic rusty sputa are observed in the course of heart disease, it is probable that the hæmorrhage is complicated by local consecutive hepatisation. This is often recognised during life, but still more often it is ascertained after the patient's death.

It is well known that children do not expectorate; hence we lose in these cases the important help derived from inspection of the sputum. Even as late as the age of eleven and twelve years the patient may be unable or unwilling to bring up the phlegm. When a child vomits after coughing we may sometimes observe characteristic rusty sputa in the basin. Old persons, as a rule, are like children in this respect; they seem unable to expectorate the sputa.

The nervous system is less frequently and severely affected in patients suffering from pneumonia than in most cases of typhus or enteric fever; but more frequently than in rheumatism without hyperpyrexia. From the beginning the patient's rest is disturbed; and if the case be protracted, insomnia may become a grave and distressing incident. Some degree of nocturnal delirium probably accompanies almost every state of pyrexia, even that which attends a feverish cold; and it is sometimes severe in attacks of influenza otherwise mild. In this slight degree, shown by the persistence of the impression of dreams, and by confusion of time and place, delirium is probably present in every case of pneumonia. It seems never to take the terrible form occasionally seen in rheumatic fever, and associated with hyperpyrexia and pericarditis; but it is sometimes important from its preventing sleep and prompting attempts to get out of bed. When an intemperate person is seized with pneumonia, the febrile delirium, as in other cases of this nature, assumes the characters of delirium tremens; and the gravity of the case is at once apparent. But even in pneumonia of the most temperate persons diurnal delirium is a serious symptom.

Termination.—In a slight case of pneumonia, particularly when affecting a child, after a sharp onset and a day or two of fever the temperature falls rapidly, the skin begins to act, refreshing sleep is obtained, and the patient awakes on the third day to find convalescence begun. These cases are, however, the exception. The symptoms much more often, even in favourable cases, last till the fifth, the sixth, or the seventh day; and in many persons who make a good recovery the illness is prolonged into the second week, so as to give a duration of eight, nine, or ten days. Beyond this the duration of the disease, unless due to some local cause, is certainly rare. The most frequent extension of the inflammation is to the other lung; but pleuritic effusion, and particularly effusion of pus, pericarditis, or severe and persistent bronchial catarrh may protract the disease beyond its natural limit. (Appendix, p. 135.)

In unfavourable cases it is very rare for death to take place on the first or second day, as in malignant variola or scarlatina. Even with double pneumonia the patient seldom succumbs before the fourth day; the time of greatest anxiety is that of the latter days of the first and the early days of the second week. In such cases, while the temperature still ranges high, the insomnia and delirium become more severe; the tongue is dry and brown as in typhus, and the pulse more frequent, weaker, and perhaps irregular. The rapidity of breathing, the dyspnœa and the cyanosis depend upon the extent of lung involved. When the whole of one lung is hepatised, the other rarely escapes; and in such cases additional difficulties in aerating the blood are often caused by profuse bronchial secretion which chokes the air-passages, passes up and down with each weak breath, and is not expelled by a vigorous cough. In the other cases, even with moderate secretion and without signs of cyanosis, the heart begins to flag, the pulse grows small and weak, and the condition becomes like that of a typhoid patient at the end of the third week.

The favourable close of an attack of pneumonia, whether earlier or later, will almost always be marked by a critical fall of temperature; and even in cases which prove fatal by some intercurrent complication, this crisis may often be observed. Indeed a gradual fall of temperature, or a temperature which continues high after ten or twelve days, should lead to a revision of the diagnosis, and to a careful search for some disturbing condition apart from the primary disease.

Physical signs.—The auscultatory evidence of pneumonia, which was discovered by Auenbruger and Laennec, and has since been confirmed, corrected, and elaborated by a succession of physicians, is in the majority of cases striking and unequivocal. It suffices for the recognition of the presence, extent, and course of pneumonia without the aid of other symptoms; just as a sure diagnosis of the disease can be made from the aspect of the patient, the pain, the sputum, the urine and the fever, without the aid of percussion or auscultation. Together they make the recognition of primary pneumonia one of the easiest of the physician's tasks.

It is seldom that we have the opportunity of examining the chest at the beginning of the disease. Within a few hours of the initial rigor and rise of temperature we often find the percussion note at the base of one lung less clear and flatter than at the other; the respiratory murmur has lost its normal character, and has assumed more or less of the bronchial, tracheal, or tubular quality. Sometimes, however, mingled with this diminished dulness and with the "vesiculo-bronchial" breathing, or even preceding it by a few hours, may be heard an adventitious murmur which is very characteristic, and was thought by the earlier auscultators to be even more frequent and more decisive than later experience has confirmed. This is the *râle crépitant* of Laennec, who briefly described it in the following words:—"Dans le premier degré de la péripneumonie, la respiration s'entend encore dans le point affecté, soit que la percussion donne en cet endroit un son mat, soit qu'elle n'indique aucune différence sensible, ce qui varie. Mais la respiration, quoique sensible dans le lieu affecté, est cependant beaucoup moins grande et moins sonore que dans les autres parties de la poitrine; elle est, en outre, accompagnée, dans l'inspiration surtout, d'une espèce de crépitation ou de râle léger, dont le bruit peut être comparé à celui du sel que l'on fait décrépiter en le chauffant dans une bassine; ce râle, que j'appelle râle crépitant, est le signe pathognomonique du premier degré de la péripneumonie. Il serait difficile de le mieux décrire; mais il suffit de l'avoir entendu une seule fois pour ne plus le méconnaître" (17, § 209).

In other places the illustrious French physician admits that the same "râle crépitant" may be heard in cases of œdema of the lung. He says: "Le cylindre, dans ce cas, offre deux moyens de reconnaître l'altération du poumon. La respiration s'entend beaucoup moins qu'on ne devrait s'y attendre, à raison des efforts avec lesquels elle se fait et de la grande dilatation du thorax dont elle est accompagnée. L'on entend en même temps, comme dans la péripneumonie au premier degré, une légère crépitation plus analogue au râle qu'au bruit naturel de la respiration. Ce râle crépitant est mois fort que dans la péripneumonie au premier degré: cependant on doit avouer qu'il est fort difficile de distinguer ces deux affections l'une de l'autre à l'aide des seuls signes donnés par le cylindre, et qu'il est nécessaire d'y joindre la comparaison des symptômes généraux" (17, § 500).

This remarkable sound is, as Laennec says, inspiratory; occasionally it may be heard with expiration also, but this is exceptional. It is a "moist" sound, or, to speak more strictly, it is an interrupted sound; the parts of which it is made up are very short, very numerous, and uniform in duration and quality. Though perfectly audible and distinct, it is not loud; lastly, its quality is thin, rather high pitched, and what is called "musical," "bright," or "clear"; that is to say, it has more tone and is farther removed from a mere noise than the respiratory murmur, a sonorous rhonchus, or than the bubbling râles in the trachea which form the death-rattle. On the other hand, it is more of a noise, and has less tone or musical quality than the metallic tinkling heard in a large cavity, or the clear percussion note of pneumothorax or of gastric distension. Unlike other râles, it is not influenced by coughing. It sometimes persists for a few hours only; sometimes it lasts until dulness and tubular breathing show that the lung is completely solid; sometimes it leaves the place where it was first heard, and ascends with the advancing line of hepatisation; or it appears on the opposite side as the first sign of extension to the other lung. Lastly, under the title "crepitus redux," it may reappear, somewhat larger, louder and less musical, when the solidified lung is recovering and again admitting the air.

This pneumonic crepitation is so peculiar and remarkable a sound that after it has been heard two or three times it is easily recognised; but, beside the account of its acoustic properties attempted above, it may be compared to some other sounds. Laennec's own illustration, quoted above, is that of the crackling sound produced by heating salt over the fire; this resembles the râle in being an interrupted sound, and in the clear sharpness of its quality; but the crackles are fewer, larger, and louder. The late Dr. C. J. B. Williams compared pneumonic crepitation to the sound heard when a lock of hair is gently rubbed between the fingers close to the ear. My own illustration for students was by squeezing a piece of the porous indiarubber, formerly much used instead of a toilet sponge, after it had been soaked in water. The late Dr. Sturges reproduced the sound by pressing dry tissue-paper into a ball, or by squeezing and relaxing a piece of sponge dipped in gum-water.

The "redux" crepitation is sometimes indistinguishable from that of pulmonary hæmorrhage, or of broncho-pneumonia, or from that which is commonly regarded, since Laennec's time, as a sign of œdema of the lung.

The true pneumonic crepitation undoubtedly differs from these in being finer or smaller, that is to say, the crackles are shorter and more numerous; they have more tone and are less loud; they are more often confined to inspiration, and are uninfluenced by deep breathing or by cough.

Nevertheless the ear of most auscultators tells them that these sounds are all similar; thus from the more definite pneumonic crepitation we pass by small gradations through "redux" crepitation to that of broncho-pneumonia, and so on to the smaller consonating râles of phthisis. On the other hand, pneumonic crepitation is quite unlike any respiratory murmur, modified or unmodified; unlike sibilus or any other continuous sound, and unlike non-consonating râles of every degree. The only sound heard in the chest which may simulate it is a very soft pleuritic rub, not loud, but clear and audible at the end of inspiration.

The physical explanations of this remarkable auscultatory sign are of doubtful validity. Probably the most generally received opinion is that it is caused by the opening out of the small extra-lobular bronchioles, by the inspired air, while their walls are covered with viscous exudation. This explanation, however, appears improbable, as it can scarcely be applied to the explanation of expiratory crepitation; and we have no evidence that collapse of the channels of the air-passages occurs as a matter of fact. Moreover, true pneumonic crepitation is thus too widely separated from the allied sounds above enumerated. From these indeed it may be distinguished by the practised auscultator, who rightly teaches students to make the same distinction; nevertheless we must admit with Laennec that the distinction is sometimes difficult. The air-vesicles cannot be the seat of this crepitation any more than they can be the seat of the normal respiratory murmur, or indeed of any auscultatory events; for the air in the vesicles is not changed save by diffusion: the strongest efforts of respiration produce a current in the air-passages only, which does not reach the lobules. Pneumonic crepitation is an interrupted and probably a "moist" sound, that is, a sound made by bubbles bursting, and is a consonating râle, that is, a sound transmitted through a solid lung. Regarding it, then, as the finest or smallest of this group, we may ascribe its production to the same cause, namely, to air passing through fluid and making a series of crackles which are transmitted to the ear through a hepatised lung.

There is little reason to acquiesce in the three stages of pneumonia described by Laennec, either on anatomical or clinical grounds. The first stage, that of engorgement or active congestion, is in all probability the beginning of hepatisation, and with the afflux of blood comes the exudation; congestion and hepatisation begin and go on together, and the consonating râle just discussed is the earliest sign, not of preliminary hyperæmia, but of actually existing inflammation. Hence we find it mingled with the signs next to be described, signs which are admitted to denote complete solidification of the lung.

These are bronchial breathing, bronchophony, and increased vocal fremitus.

This is not the place to discuss the difficult questions involved in the physical theory of the respiratory murmur and its modifications in disease. It must suffice to assume, as most in accordance with physical facts and least contradicted by clinical experience, the hypothesis which refers the normal respiratory murmur to vibrations of air due to a fluid vein, which is produced in inspiration and expiration by the passage of the air through the narrow chink of the glottis into the wide channel of the trachea in the one case, and of the upper larynx in the other. The sounds thus produced are heard by a stethoscope placed upon the larynx or trachea; where they have the loud, somewhat harsh, continuous, blowing character, which is recognised as tracheal, or an extreme degree of bronchial or tubular breathing. On listening to the patient's voice with a stethoscope on the trachea we hear the loud vocal resonance which is known as bronchophony. The same inspiratory and expiratory murmur and vocal resonance are heard in most persons over the manubrium; and in some on applying the stethoscope between the shoulder-blades, or over the inner part of the first right intercostal space: but the sounds thus transmitted (not by the column of air, but by the walls of the trachea and right bronchus), while still harsh and blowing, are less loud than the tracheal murmur just described. This bronchial breathing is certainly no new sound; it is the tracheal murmur diminished in intensity, because less directly conveyed. As soon as we leave the neighbourhood of the trachea (that is to say, over nearly, if not quite, the whole of the chest) we find the resonance of the voice much diminished, even when it is that of a strong man with thin covering of the thorax; while if the thorax be thickly covered by clothing, subcutaneous fat, or even thick muscles, or if the voice, as in women and children, be shrill and comparatively feeble, the vocal resonance is weak and not infrequently absent.

Since the vibrations of speech in the larynx are conducted by the walls of the air-passages, and when they reach the surface of a healthy lung are still the same sounds, though diminished in loudness or modified in character; and since again the sound of the voice is greatly altered when transmitted through a solid lung, or through pleural effusion, or through emphysematous lung, or through pneumothorax, yet, however altered or modified, is still the sound produced by the vibration of the vocal cords —so, according to the hypothesis now advocated, the breath-sounds heard over the chest are not produced in the pulmonary vesicles, the lobules or the bronchial tubes, but in the larynx.

The pulmonary murmur is heard indeed over the lungs, but does not arise there. It is the same sound which over the trachea we call tubular, with its loudness diminished and its quality altered by transmission through the spongy lung.

On this view we do not start with a so-called vesicular murmur in the lung, which becomes bronchial in the bronchi and tracheal in the larynx; but in breathing and in voice we regard the audible vibrations as formed at the glottis and altered, whether in health or disease, by transmission through various media.

Now when, as in pneumonic hepatisation, the laryngeal breath-sounds are transmitted through a solid lung, they retain much of their loudness and quality. The expiratory murmur is not shortened and weakened, or even rendered inaudible, as in health. Inspiration and expiration are accompanied by a murmur nearly equal in length, loudness, pitch and quality.

The simplest way of describing the breath-sounds heard in pneumonia is to call them tubular, bronchial, or tracheal; and to define them by reference to the respiratory murmur as heard over the trachea or the manubrium in health. But, simple as it is, this comparison is not completely accurate; it may help the student in the rudiments of auscultation, but it will probably hinder him when he hears well-marked pneumonic breathing. It seems that in phthisis, lobular pneumonia of children, and other conditions of partial solidification, and again in some cases of narrowing of a large bronchial tube whether by stricture or compression from without, breath-sounds may be heard which are very different from the pulmonary murmur, and which closely approach that heard over the larynx. These may be fairly called bronchial or vesiculobronchial sounds. They differ from the tracheal murmur chiefly in loudness, so that expiration loses its later part and becomes shorter than when heard over the larynx, though longer than over a healthy lung.

The word "tubular" is often applied indiscriminately as a synonym of tracheal or bronchial breathing; but by many writers, and I think with advantage, it is reserved for the special modification heard most perfectly in cases of pneumonic hepatisation.

This modification essentially resembles laryngeal, tracheal, and bronchial breathing, and differs essentially from the healthy pulmonary murmur. It has a blowing rather than a breezy quality; and expiration is often as loud and usually as long as inspiration; but it differs from the normal laryngeal murmur in the following points: it is not so loud, nay, it may be softer than the bronchial breathing above described; but, however subdued, it is remarkably distinct—audible, that is, by reason of its peculiar quality, when a much louder breath-sound might pass unnoticed. Again, it is higher pitched than the pulmonary murmur and than laryngeal or tracheal breath-sounds; thirdly, it has the quality of tone, that is to say, it is farther removed from a mere noise, and approaches though it never reaches the quality of a musical note. To these characteristics we may add that expiration does not follow inspiration so immediately as in health—possibly because the solidified lung does not contribute its resilient energy to that of the rest of the lungs, or it may be because of some disturbance of the nervous mechanism of respiration. In short, the laryngeal vibrations in breathing are transmitted to the ear better than in health, less changed and changed in another direction.

Along with the tubular breathing marked bronchophony is present, that is, when the patient either speaks or coughs, the laryngeal voice is heard more loudly and distinctly than with a healthy lung. The tactile fremitus is also more perceptible, that is, the laryngeal vibrations are more perfectly transmitted to the touch as well as to the ear.

A remarkable variety of vocal resonance usually heard over a pneumonic lung is that called by Laennec "pectoriloquy"; in which case not only the laryngeal voice but also the articulated sounds in the mouth are transmitted, so that words as well as vocal sounds are heard. If the patient speak in a whisper, the latter of course are absent, and we hear the former alone, just as if a loud whisper were directed into the stethoscope. Thus pectoriloquy is best distinguished as whispered pectoriloquy, but vocal pectoriloquy is just as real a phenomenon, and was what Laennec first described.

There are some curious exceptions to the regularity of these physical signs. Occasionally the percussion note loses its resonance, but at the same time rises in pitch; this condition, sometimes found over a very tense coil of intestine, is known as tympanitic dulness. Still more rarely a solid and hepatised lung has been found by good observers to yield a resonant note, even in the presence of bronchial breathing and bronchophony. Occasionally, instead of the ordinary dull or flat toneless percussion note, a "cracked-pot" sound may be heard, particularly in children.

In some cases, where presumably there is a plug of fibrinous exudation, bronchial breathing and the associated signs are absent: such a case resembles a pleural effusion save that the heart is not displaced.

Since pneumonia is always accompanied by pleurisy, a pleuritic rub will almost always be heard early in the attack; most often to the outer side of the nipple or near the angle of the scapula.

The physical signs just described undergo little alteration as during the progress of the disease hepatisation spreads upward, or attacks the other lung: pneumonic crepitation may be heard at the advancing border, and the area of dulness and tubular breathing extends. When the crisis arrives, the solidified lung clears up, but more slowly than the symptoms. The dulness gradually gives place to resonance; the "redux crepitation" is often heard, and instead of tubular breathing we hear râles, at first consonating and then losing this quality as they become larger and looser.

Sometimes the signs of consolidation continue for a week after the temperature has become normal, or even longer than this. Sometimes they are succeeded by the signs of pleural effusion.

Diagnosis. — It is customary, after describing the symptoms and signs by which a disease is recognised, to add special directions for distinguishing it from other diseases. But it is often impossible to predict what maladies may be confused in a given case; and when we draw up tables of distinguishing signs, although the exercise is useful for a student, we find, on the one hand, that each has to be so guarded and qualified that it ceases to bear the weight put upon it, or on the other that we are making our statements more absolute than facts will bear out: thus we may mislead rather than help the reader. There are no pathognomonic symptoms of any disease, nor is there any royal road to diagnosis.

In some cases, as in the discrimination of external tumours, in the distinction between measles and rubeola, rheumatism and gonorrhœal synovitis, psoriasis and scaly syphilis, tuberculosis and enterica, it is practically useful to keep the contrasted features in our minds; though in each of these cases we may rely too much upon them, and may fail to observe other points of greater importance. But pneumonia is so well marked a malady in its symptoms and course, and auscultation gives us such clear and precise evidence of its presence, that, once suspected, it can always be discovered; and the disease is too common and striking not to be thought of whenever an acute febrile attack is before us.

In children, confluent pulmonary catarrh may simulate lobar pneumonia in its physical signs; but its onset and course are very different, so that we have only to bear in mind that true pneumonia may affect young children and even infants at the breast.

Apical pneumonia has been mistaken for phthisis; but if the sputa and the curve of temperature do not distinguish them, the onset and the crisis are decisive.

When pneumonia comes on in its secondary form as a complication of some chronic disease, it may be easily overlooked; but, if sought for, the characteristic physical signs prevent all doubt of its presence. A rise of temperature, with rapid breathing or quickly ingravescent muscular weakness, are the symptoms that should at once lead to auscultation of the lungs. When pneumonia complicates fevers we are already on the watch, and an increased ratio of respiration to pulse and temperature at once excites suspicion and indicates the danger. In the course of delirium tremens we must always be on the look-out for the supervention of pneumonia; for the entire lower lobe of a lung may be hepatised before the appearance of characteristic symptoms.

In children, fever, dyspnœa, and cough may coincide with dulness on percussion and characteristic tubular breathing at the base of one lung, and the cause may be not hepatisation, but pleural effusion, most often purulent. No sputum is obtainable, and the voice fails to give help. In such cases, and in such only, we are driven to leave the art of diagnosis by physical signs, and to solve the problem by puncture with a hypodermic syringe.

Other difficulties of diagnosis turn rather upon disputed pathological points than on technical skill in interpreting symptoms and signs.

The first depends upon the distinction of true pneumonia from what has been often described as "acute pulmonary congestion." This affection, described by practitioners in England, and admitted by some pathologists in France, may be defined as an acute disease with the symptoms but without the physical signs of pneumonia, running a short and for the most part a benign course, usually calling for and sometimes obtaining antiphlogistic treatment. That an attack of pneumonia occasionally aborts, that the physical signs of pneumonia are sometimes delayed for two or three days after the symptoms have appeared, and that they are sometimes strangely obscured or difficult to detect, are facts that probably explain most of these cases. The remainder may perhaps be regarded as examples of acute pulmonary œdema, like that which not infrequently attends the later stages of Bright's disease. In any case acute arterial congestion without exudation — hyperæmia without inflammation — is a pathological event the existence of which has never been proved, and cannot be admitted until supported by the results of post-mortem examination.

Another question of diagnosis depends upon our view of the relation of lobar and lobular pneumonia in children. When patches of catarrhal pneumonia become confluent, the physical signs closely resemble those of fibrinous hepatisation; the symptoms are also more acute in these cases, and may be undistinguishable from those of the latter. Even anatomically it is so difficult to distinguish them that some good pathologists give up the attempt. Moreover, the pneumococcus may be present in lobular as well as in lobar pneumonia. The distinction rests on the more gradual onset, the relation to previous bronchitis, measles, and other primary disease, the more scattered localisation; anatomically, on the separate lobules being distinguishable, the surface less firm and granular; and microscopically, on the greater amount of leucocytes and epithelium, and the smaller amount of fibrin and blood-discs. Confessedly difficult as is the diagnosis between true lobar pneumonia and lobular catarrhal consolidation, there does not appear to be sufficient reason for giving up the attempt. The two diseases differ essentially in their natural history, origin, incidence, course, and histology; and the difficulty of distinguishing them in what is after all a small proportion of cases is no more a reason for confounding them than in the similar occasional difficulty of distinguishing between rubeola and morbilli, osteoarthritis and gout, carcinoma and alveolar sarcoma. If the whole duty of medicine were the practical one of healing the individual patient, we might be content with the knowledge that in the doubtful cases, which sometimes occur in children, the determination of the question is not of practical importance.

The third difficulty in diagnosis is between pneumonia with hepatisation of the lung and the cases of acute pleurisy and even of acute meningitis, cerebral or cerebro-spinal, of acute pericarditis, or ulcerative endocarditis, which, as it is asserted, have the same general symptoms and course, the same infective microbe, the same origin and pathological nature, and yet no affection of the lungs—pneumonic fever without pneumonia.

The diagnosis in these cases would rest upon the absence of the physical signs of a solidified lung and of rusty expectoration and other strictly pulmonary symptoms. The evidence for the theory in question will be noticed again in the section on pathology.

Complications and Sequels.—Pneumonia is more uniform in its course than most febrile disorders, and offers in this, as in other respects, a marked contrast to enteric fever. Its complications are few and rare.

Hyperpyrexia is comparatively rare. The temperature runs high, but seldom exceeds 106°; and deaths from this cause alone are uncommon. At the same time a temperature above 104° marks a severe attack, at any rate in an adult.

Pleurisy is not a complication, but part of the disease. In its usual fibrinous form it is insignificant except for the pain it causes. Serous effusion is seldom considerable, and rarely needs attention; but after the pneumonia has subsided, it sometimes happens that the dulness persists longer than usual, and the temperature rises again after the initial fall —the interval in Dr. White's 26 cases was from one to four days.[1] This almost always points to the presence of fluid in the pleural cavity, and as a rule this fluid is purulent. In a case lately under my own care, the pus, when drawn off, was found to be a pure cultivation of Fränkel's pneumococcus; and this is frequently the case. But more often the organisms found in the effusion are the strepto- or staphylo-cocci of non-specific suppuration. It is certainly remarkable that the diplococcus, which usually produces non-suppurative inflammation of the lungs and pleura, should occasionally cause the purulent infiltration which marks the last stage of hepatisation, and the empyema which we are considering: the two conditions, though so far comparable, seldom or never exist together; a mixed infection might rather have been anticipated, pyogenetic microbes being added, as in the latter stages of enteric fever, to those which are specific. On this point further evidence is needed.

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[1] In a paper in the *Guy's Hospital Reports* for the past year (vol. li.) by Drs. Hale White and A. C. Pearse, 26 cases of empyema are recorded following 890 cases of lobar pneumonia. The percentage on the total is three, but in different years it varied widely from none up to more than five per cent. Of 708 cases of pneumonia recorded in the *St. Thomas's Hospital Reports* (vol. xix.), only 6 were followed by empyema.

From the clinical point of view it seems very doubtful whether time will justify the belief entertained by some French pathologists, that an empyema of diplococcous origin is benign, and may be absorbed without surgical interference, while the contrary is true of one in which streptococci are found. In the case above mentioned a second paracentesis was necessary, and ultimate incision and drainage before recovery ensued.

A common and more dangerous complication is pericarditis, which, when it complicates double pneumonia, is most often fatal. It occurs during the height of the fever, and is recognised by the usual friction sound, which must be distinguished from a pleuritic rub produced by the impulse of the heart. The effusion is usually plastic and serous, but occasionally it is purulent. There is no added pain, but dyspnœa and orthopnœa are increased, and the pulse becomes smaller and irregular.

Another complication or sequel is ulcerative endocarditis. Dr. Osler, who in 1885 drew attention to this connection of the two diseases, met with it sixteen times in 100 cases. This was probably an accidentally high number. In the 425 cases tabulated by myself it occurred seven times. It often accompanies empyema.

A rarer complication is acute meningitis. Of this I have had but small experience. In the few cases I have seen, the pneumonic diplococci were found in the lymph at the base of the brain; and this appears to be the rule. Meningitis is often associated with acute ulcerative endocarditis.

Dr. Bristowe and other writers have described a membranous catarrhal or ulcerative colitis as complicating acute pneumonia. There have been several cases at Guy's Hospital during the experience of many years, but I have myself only once found this condition of ulceration of the colon. It is certainly far more common in cases of Bright's disease.

Severe epistaxis occasionally marks the onset of pneumonia, and may recur during its course. Except in elderly patients, this is not a serious symptom.

A curious occasional complication of pneumonia is jaundice; and this is more often observed when the right lung is affected. None of the attempted explanations of its occurrence is satisfactory. There is no evidence of catarrh of the bile-duct (if catarrh is ever the cause of icterus), there is no reason to suppose the blood differently constituted in these particular cases (if there be such a thing as hæmatogenous jaundice), the hepatic circulation is not more obstructed in these cases than in others without the symptom in question, and deficient movement of the diaphragm, if a cause, would be more likely to induce jaundice in cases of right hydrothorax or phrenic pleurisy. It does not appear to affect the course of the attack or its prognosis.

Relapse after recovery from pneumonia has been often reported; and cautions are given to guard against it. But I have never met with a case, and believe that a relapse occurs very rarely, if ever. The spread of pneumonia to the other lung when it had subsided in the first, or the supervention of empyema, may perhaps have been thus misinterpreted.

Recurrence of pneumonia after several months or a year's interval is, however, often observed. In the 425 cases tabulated, such recurrence is noted in eighteen. This shows that pneumonia does not protect against a future attack like small-pox or measles, not perhaps that it disposes to a repetition like erysipelas; occasionally the same patient may suffer five or six times from pneumonia in his lifetime.

SECONDARY PNEUMONIA.—The description of the symptoms and course of pneumonia above given refers with comparatively small variation to cases of primary, or, as we say, idiopathic pneumonia; to cases, that is, in which the disease, whatever its cause, is not a complication of any previous malady, but attacks a person in apparent health. There are, however, numberless cases in which after death we find lobar hepatisation as the closing event of preceding disease, acute or chronic. The anatomy to the naked eye is the same; and neither histological nor bacterial investigation enables us to distinguish the forms of primary from those of secondary pneumonia; or to distinguish one from another those which occur in the course of typhus, rheumatic fever, diabetes, or Bright's disease.

It would be an arbitrary proceeding to separate the one group of cases from the other: for, in the first place, the anatomical changes of disease are by far the safest guide in their discrimination and recognition; and, secondly, although the symptoms of these secondary forms of pneumonia are less clear and distinct than in the primary cases, yet they are present, curtailed or obscured it may be, but capable of detection, and accompanied by the same physical signs which denote the presence of primary pneumonia.

Moreover, we find a connecting link between primary and secondary pneumonia in cases following influenza, which in other respects have the character of the idiopathic disease rather than that of a complication or sequel.

When one disease supervenes upon another, the former is not likely to preserve the distinctive features which mark its invasion of a normal organism. The contrast between health and disease is more obvious than between one kind of sickness and another. The antecedent disorder of the pulse, temperature and respiration, of the appetite, of the blood, of the tongue, and of the skin, blurs the outline of those striking changes which in a healthy subject denote the advent of pneumonia. In like manner after death we may discover the ulceration of enteric fever which has supervened in a course of protracted rheumatism with relapses; or cancer in the body of a patient who has died from alcoholic intemperance; or Bright's disease in a case of emphysema and bronchitis; or phthisis latent in a diabetic or insane patient.

The diseases in which secondary pneumonia frequently occurs as a complication are the following: typhus, enteric and relapsing fevers, small-pox, erysipelas, puerperal septicæmia, and occasionally diphtheria.

Lobar fibrinous pneumonia, of which alone we are now speaking, is rarely seen as a complication of measles, but it is less rare in scarlatina. It seldom appears in the course of phthisis, or again of quinsy, bronchitis, pleurisy, or asthma. It may be seen as a complication of cardiac disease, but is much more frequent in both the acute and chronic form of Bright's disease. It is an occasional and often fatal complication of rheumatic fever; and is almost invariably fatal when associated with pericarditis. Acute lobar pneumonia of the base is often found as the cause of death in diabetes, and in chronic alcoholic poisoning; and it may occur as a fatal complication in the course of many chronic diseases, such as paraplegia, tabes, or cancer. Pneumonia, probably of septic origin but neither lobular nor suppurative, is also not an infrequent cause of death after surgical operations. It was remarked by the great Dr. Arbuthnot, in the 3rd chapter of his book on Diet, that a peripneumony is the last phase of every disease; for nobody dies without a stagnation of the blood in the lungs. If we include broncho-pneumonia and hypostatic congestion under the name of pneumonia, as Arbuthnot, no doubt, would have done, the proposition is not very far from the truth.

In what points do these secondary forms of pneumonia differ from the primary? In the first place, they are far more dangerous. Secondly, although their anatomy and their physical signs are the same, they lack some of the symptoms of the idiopathic disease: more particularly it may be observed that the initial rigor is frequently, perhaps usually, absent; that the temperature does not rise so abruptly or so high; that although pleurisy is present, and a rub may be heard if sought for, yet pain in the side, instead of being a constant symptom, is often absent; that a cough may also be wanting; that the burning heat of the skin and the characteristic conditions of the urine may be absent or slightly marked. Herpes is usually absent. The sputum is rusty, and contains diplococci; but often in these cases, either from absence of bronchial irritation or from the weakness of the patient, no sputum is expectorated. If all these symptoms are wanting, and fever already present, a secondary pneumonia may be called latent; and we have then to depend entirely upon physical examination of the chest for its recognition. Apart from these signs, probably the most important symptom is increased frequency of respiration.

Other clinical varieties.—Beside the distinction between primary and secondary pneumonia, certain other divisions of the disease have been made, by some writers, into varieties which scarcely deserve separate nomination.

Abortive or *ephemeral* cases, which last only a day or two; wandering pneumonia (*P. migrans*), in which form the disease attacks first the right base, then the left, and then perhaps returns to the right apex; *malarial* pneumonia, in which under the influence of this poison the fever assumes an intermittent character, are among the varieties which might be multiplied without advantage.

In children, pneumonia is comparatively rare, its course usually short, and its prognosis very good. The pneumonia of old people is often attended by only moderate fever; bronchitis is common and exhaustion speedy. The drunkard's pneumonia is complicated with early delirium.

Epidemic pneumonia, apart from that consecutive to influenza, has been frequently observed in former and recent times. An interesting historical account of it is given by the late Dr. Wilson Fox (12) (13), and also in Sturges' and Coupland's valuable monograph. Recent epidemics have been described by Dr. Whitelegge in the first volume of the present work (p. 655).

Morbid anatomy.—The anatomy of pneumonia is no less striking and characteristic than its symptoms or its physical signs. The hepatised lung does not collapse on exposure to atmospheric pressure. It feels heavy when lifted out of the chest, and readily sinks in water. Its surface is covered by a more or less extensive layer of fibrin. This false membrane, or coagulated lymph as it used to be called, is sometimes a thin, adherent gray film, contrasting with the smooth and shining portions of the membrane which are unaffected. Sometimes it is in thick and comparatively tough layers, which can be peeled off; sometimes in soft shaggy masses of moist fibrin; and sometimes, together with the solid exudation, there is more or less of serum or pus.

On cutting into the lung the section is seen to be dark red, the colour of liver; or at a later stage a pale and yellowish gray. The advancing border is red and œdematous, but not yet solid. The surface is granular, uniform, and dry compared with most other morbid states of the lung. As in other acute inflammations, the tissue is so softened that it readily breaks down under pressure. A scanty, blood-stained liquid, characteristically free from froth, issues from the squeezed tissue; and this becomes more abundant, paler, and thicker in the parts longest hepatised —that is to say, in the gray as compared with the red hepatisation.

The traditional stages of pneumonic hepatisation are—(1) Engorgement (Bayle), that is, acute congestion with œdema, but without solidity; (2) Red hepatisation, that is, solid exudation with hyperæmia; (3) Gray hepatisation or solid exudation with anæmia from pressure on the blood-vessels; (4) Purulent infiltration, the gray tissue yielding thick purulent exudation.

Histology.—On scraping the cut surface we obtain a turbid liquid, of which a drop under the microscope shows abundant red blood-discs, with a few leucocytes; or, in the latter stages, abundant leucocytes, and a few blood-discs, together with minute fibrinous casts of the vesicles. Larger branching casts, plainly visible to the naked eye, can often be pulled out of the bronchioles with forceps. Diplococci may be detected after staining, and sometimes streptococci in addition.

A thin section of the hepatised lung shows the vesicles and intralobular air-passages, as well as the smallest bronchial tubes, to be completely filled with exudation: blood-discs, threads of fibrin, and leucocytes, the latter at first few in number, but becoming more numerous as the red turns to the gray stage.

The absence of epithelial cells is important. The walls of the air-vesicles are usually thickened, but no other change of the texture of the lung is apparent, no alteration of the elastic fibre, and no increase of the exceedingly scanty interstitial connective tissue of the healthy lung. The capillary blood-vessels in the early stages of hepatisation are dilated; but the blood is gradually squeezed out of them as consolidation goes on until red gives place to gray hepatisation, when the lung becomes very anæmic. The remarkable friability of a pneumonic lung is caused partly by the swelling and softening of the tissue, but chiefly by its being solid instead of spongy, so that instead of yielding to pressure, it resists and breaks down under it. In the later stages the blood-discs are no longer seen, or only few of them; the leucocytes, on the other hand, have greatly increased in number, and have assumed the granular character of pus-cells. The air-vesicles, being thus stuffed and swollen with inflammatory exudation, expel the blood from the pulmonary capillaries and cause the exsanguine pale aspect of gray hepatisation. The gray colour is due to the accidental pigmentation with granules of carbon due to inspiring smoky air, and is absent when the hepatisation of the lungs is observed in children or in cattle.

Anatomical distribution.—The site of hepatisation is important. In the majority of cases (288 : 92) it begins at the base of the lung and travels slowly upwards—in the reverse direction to that characteristic of phthisis. The epithet lobar is to some extent unfortunate; for the process is seldom exactly bounded by the fissures of the lung. The lower lobe may be hepatised only in its posterior-inferior part, or in all but its apex; or the back of the upper lobe and the whole of the middle and lower lobes of the right lung may be solid and the apex alone escape. Sometimes every part of one lung is found solid after death; the base gray and the upper parts red, with hepatisation of the base of the other lung.

Not infrequently, however, pneumonia attacks the apex and travels downwards. This local variety is more common in children than in adults; it is often latent, and is said to be more often associated with delirium. The prognosis of apical pneumonia, when it occurs in an adult, does not seem to be better or worse than that of the ordinary basal form.

Apical pneumonia of one lung is often accompanied by basal pneumonia of the other. Double apical pneumonia is very rare.

A third site, still more seldom selected by the pneumonic process, is the deep part of the lung near its root. This "central pneumonia" is naturally difficult of detection before it has advanced towards the surface.

Occasionally the anterior tongue-like process of the left lung is alone affected on that side.

Right pneumonia is rather more frequent than left, whether it affect base or apex,[1] and unilateral is happily more common than double pneumonia. (See Appendix, § 4, p. 132.)

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[1] It happens that in the cases collected by the writer there were rather more of the left than of the right base (151 : 137), but in larger numbers the proportion is reversed.

Anatomical events.—In the majority of the cases pneumonic inflammation ends by resolution. The exuded blood-discs, and leucocytes, and fibrin are no longer expectorated as such, but degenerate and are broken up into granules, chiefly fatty in nature. These are partly mingled with the bronchial secretion and coughed up, but probably a larger proportion are absorbed by the lymphatics, conveyed to the veins, and finally excreted. The abundant salts (chiefly sodium-chloride) deposited in the inflamed lung are rapidly carried off by the veins, and discharged in the urine. The air re-enters the minute bronchi and air-vesicles as they re-expand in inspiration. The lung more or less rapidly regains its spongy character, and the pleural exudation is gradually absorbed. Once only have I had the opportunity of seeing a hepatised lung in process of recovery about ten days after the crisis; it was a case in which death occurred from another cause. The tissue contained air, exuded frothy serum, and floated in water; it was anæmic rather than congested, did not break down under pressure, and was very œdematous. As above stated, we learn from physical examination of the chest that the hepatised lung sometimes continues solid for several days after the raised temperature and other febrile symptoms have disappeared.

In fatal cases we may find any stage of hepatisation, as above described, and any combination of these stages. There is no reason to conclude that gray consolidation, or even purulent infiltration, is incapable of resolution; but the contrary opinion is obviously almost as difficult to prove as to disprove.

When the lung has passed into gangrene the surface is obscured by a cloudy film; the tissue breaks on the slightest handling, is of a very dark colour, and emits a characteristic foul and pungent odour. Under the microscope no air-vesicles or other structural forms are distinguishable; the organ and the exudation are alike dead and disintegrated, and blood-vessels and elastic tissue alone remain. Gangrene is always limited in extent, but perhaps never by a separating capsule. In many cases, probably in most, the cause of the gangrene can be ascribed to the presence of particles of decomposing food which have gained an entrance to the air-passages in the last day or two of the patient's life.

It has been stated by Addison, and by other pathologists, that occasionally a pneumonic lung never recovers its permeability to air, and may gradually pass into an unnaturally firm, pale, solid state, containing an excess of fibrous tissue, a state which has been included by some writers under the head of cirrhosis. Addison described it as "marbled induration." This condition must be a very rare one, and it does not appear to lead to the contraction of tissue or the dilatation of bronchi which mark Corrigan's cirrhosis.

Equally supported by credible authorities, both past and present,[1] is the statement that pneumonic hepatisation may end in abscess of the lung. Of this, as of the previously mentioned condition, I have no experience, and without denying the occurrence of either, would regard this also as an extremely rare event. The "abscesses of the lungs" of the physicians of the 17th and 18th centuries were tuberculous vomicæ. Multiple pyæmic abscesses, now happily seldom seen, are well-known morbid conditions, but they do not follow true pneumonia. A small circumscribed empyema following pneumonia may burst and simulate an abscess during life, and may even be misinterpreted after death. And when the stage of purulent infiltration has been reached, it is not difficult by pressure or accidental laceration of the rotten tissue to produce a cavity filled with purulent fluid not at all unlike an abscess. These facts are, however, familiar to the able pathologists who describe abscess as a not infrequent event of (acute, lobar, fibrinous, or "croupous") pneumonia, and therefore its occurrence cannot be denied: but subsequent experience has certainly confirmed the observation of Laennec—"Il n'y a pas de lésion organique plus rare qu'une véritable collection de pus dans le tissu pulmonaire" (22, § 192).

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[1] Among the former may be mentioned Stokes, among the latter Osler.

Other organs.—In an autopsy on a case of primary pneumonia we expect to find the lungs only diseased, and an exception is rare. There will always be lymph on the visceral pleura, and sometimes pleuritic effusion, serous or purulent. The bronchial lymph-glands are soft and swollen. Occasionally the pericardium or endocardium, the meninges, larynx, or large intestine may show the lesions above described.

The blood in pneumonia when drawn coagulates slowly, and forms an abundant and firm clot with the "buffy coat," due to the red discs having subsided before becoming entangled in the meshes of the fibrin as it forms. This condition (so-called hyperinosis), like the pneumonic pulse and fever, is present in many other inflammatory diseases. The leucocytes of the blood are also considerably increased in number, a fact first observed by Piorry, confirmed by Virchow, and more recently by Billings.

Bacteriology.—Klebs, in 1877, was the first to describe a microbe characteristic of pneumonia; but it was probably not the same as that afterwards discovered by Friedländer, and certainly not the same as Fränkel's diplococcus. The former was described by the late Dr. Friedländer in 1882 as an oval body 1 μ in length, occurring in pairs (*diplococcus*) or in chains (*streptococcus*); and, as was soon after ascertained, enclosed in a thick transparent envelope. On cultivation in gelatine it forms a colony of a characteristic nail-shape. When inoculated it produced pleuro-pneumonia in mice, and often in guinea-pigs, but not in rabbits. Accordingly it was named *Pneumococcus* (15).

It was soon ascertained, however, that this organism is not a coccus, but a bacterium or bacillus; that it is not invariably present in pneumonic lungs or sputa; and that it is often to be found in the mucus of the nose and bronchi, in the absence of pneumonia.

In 1884 Dr. A. Fränkel brought forward another claimant to the distinction—a microphyte, also oval or lancet-shaped, also occurring in pairs, also encapsuled, but differing in the conditions and results of successful cultivation. It also produced death in rabbits (but not pneumonia), and pneumonia in mice and guinea-pigs (14). The same microbe, in all likelihood, had been independently detected in hepatised lung by Talamon (30); and this again was the streptococcus observed by Dr. Sternberg of the U.S. Army in 1885, named by him "Micrococcus of Pasteur," and identified by many observers with that of sputum-septicæmia, and with a similar organism occurring in healthy human saliva—"*Bacillus sputigenus*," "*Bacillus salivarius*" (26).

The extended researches of Weichselbaum in Vienna on 129 cases of pneumonia (in the wide sense of the term) resulted in the discovery of Fränkel's diplococcus in 94, of Friedländer's "pneumococcus" or "bacillus-pneumonic" in only 9, streptococcus pyogenes in 21, and staphylococcus pyogenes aureus in 5 (33). This appears to show that while one microbe is most frequently present, others are competent to produce "pneumonia"; but, unfortunately, it is not clear what precise kind of pulmonary disease was present in the several instances.

The experiments of Gamaleia in Pasteur's laboratory led him to believe that the pathogenetic organism is to be found in the diplococcus described by Fränkel (now often called pneumococcus, in succession to the title enjoyed for a short period by Friedländer's bacillus), which, however, he identified with that of Talamon and Sternberg, and calls "Streptococcus lanceolatus Pasteuri" (16).

Lastly, we must remember the mobile rod-shaped microbe which was found by Dr. Klein (20) in the epidemic pneumonia of Middlesborough.

While recognising the interest and importance of these laborious researches, we must observe that even the diplococcus of Talamon and Fränkel does not fulfil Koch's three tests of a pathogenetic organism; as they are fulfilled, for example, in anthrax, relapsing fever, and tuberculosis. It is not invariably present in the tissue of pneumonic lung; it does occur in other situations in health; when injected as a pure cultivation it does not always reproduce itself and cause a fresh case of hepatisation of the lungs. Nevertheless the frequency of its occurrence, and the fact that it often reproduces the disease by inoculation, make it probable that it plays an important part in the natural history of pneumonia.

There are, indeed, two other and preliminary postulates which are no less important than the three of Koch, but which have not always been kept in view in these or in similar investigations.

The first is that the microbe of which the action is under investigation shall be "specific"—that is, it shall be possessed of definite and constant characters; it must be a good botanical species which can be identified beyond dispute. The mere shape is admitted to be illusory. The same microphyte varies in different stages of its growth and under different conditions, and may almost arbitrarily be described as a micrococcus, a diplococcus, or a streptococcus; as an oval or lancet-shaped bacterium, or as a bacillus. The presence of a capsule is not a constant distinction; and even the reaction to staining agents and the shapes assumed under various methods of cultivation are not always decisive.

But if a certainty that different observers are discussing the same microbe is essential before their results are compared, it is no less essential that they should be agreed as to the disease which is under investigation. Septic pneumonia, lobular broncho-pneumonia, and lobar fibrinous ("croupous") or vesicular pneumonia are different pathological conditions; and unless they are carefully discriminated, statistics of "inflammation of the lungs" are as useless as would be similar facts with regard to "inflammation of the joints" or "inflammation of the skin."

Pathology.—Looking back at the natural history of the disease now described, to what conclusion concerning the nature and essential characters of pneumonia and its relation to other disorders are we led? Are we, like Laennec and his successors, to regard it as an acute inflammation of the lung, of which the pyrexia and other symptoms are only the effects? Or are we, with most modern writers, to look on the local lesion as but one element in a specific infective fever? Or is any alternative opinion open to us?

In the first place, the attempt, which has more than once been made, to disprove the inflammatory nature of pneumonic hepatisation has certainly failed. We have the hyperæmia and softening of the tissue which are the characteristic signs of acute inflammation everywhere; we have the pyrexia and other febrile symptoms which also attend acute inflammation; we have the exudation of liquor-sanguinis, with leucocytes and fibrin; the last stage of the process is purulent infiltration of the affected lung, and, lastly, in every case of pneumonic hepatisation we find the obviously related pleurisy, which no one can deny to be inflammation.

It is true that pneumonia cannot be produced by injury, or by ordinary irritants, as is proved by experience in men and experiment on animals. The few supposed cases of pneumonia of traumatic or irritative origin are mostly accidental coincidences. It does not arise by extension from capillary bronchitis, nor by the irritation of dust, nor by the more specific stimulus of the tuberculous microbe; but this only shows that the process is a special, peculiar, or, as we say, specific kind of inflammation. Much the same is true of the tubal nephritis of acute Bright's disease, which is no doubt inflammation, but inflammation of a peculiar kind; not traumatic, or irritative, or septic.

Admitting, however, that pneumonia is truly inflammation of the lungs, are all its symptoms due to this local inflammation? This cannot be maintained, for occasionally we find the symptoms precede the signs of hepatisation by several days; the temperature and even the number of respirations fall when the crisis comes, long before the solidified lung has cleared up; the violence of the fever is not in proportion to the extent of the local lesions, although the amount of lung involved does produce its direct effects on the heart.

On the other hand, the differences between pneumonia and the group of diseases to which typhus, small-pox, and measles belong must not be overlooked. It resembles them in its sudden onset, in its pyrexia, its definite course, and its limited duration; but in the vast majority of cases it is sporadic and idiopathic. It does not in common experience arise from previous cases, nor produce fresh ones; it does not protect from future attacks, but rather disposes to them. Although epidemics of pneumonia undoubtedly occur, they are local, and the most striking cases are those which follow influenza, a true specific fever, just as nephritis follows scarlatina. Moreover, unlike the local lesions of enteric fever or of mumps, hepatisation of the lungs is found in the course of other fevers, and of chronic affections such as Bright's disease. Against these broad differences the fact of a distinctive microbe being present is inconclusive; for, not to insist on the absence of any such microbe in some of the most definite specific fevers, its presence is not constant in pneumonia. It is associated with other microbes, strepto- and staphylococci, and is sometimes replaced by a bacillus which Dr. Klein found constant in an epidemic of pneumonia at Middlesborough in 1889 (vol. i. p. 658). The diplococcus, which is almost constantly present, is sometimes found in lobular pneumonia, sometimes in pleurisy without pneumonia, and sometimes in healthy saliva.

The fact seems to be that in this case, as in so many others, the phenomena of disease cannot be fitted into current classifications.

Inflammation itself is a much more variable condition than it was once thought to be. It differs as the cause which produces it, and as the organ or tissue which reacts to the irritant. Bacterial diseases differ also among themselves. The constancy of the presence of a peculiar bacillus in phthisis is most important, but phthisis remains a very different disease from tuberculous meningitis, or pulpy disease of the knee; while diphtheria, erysipelas, gonorrhœa, and relapsing fever differ in almost every particular except in respect of a specific microbe. At present our knowledge is too imperfect to decide the true nature, origin, and pathology of pneumonia; but we can affirm that, on the one hand, it is not an ordinary inflammation produced by injury, or mechanical irritants, or cold, with symptoms directly proportionate to the extent of the tissue inflamed; and that, on the other hand, it differs from such specific fevers as measles, enterica, and small-pox in that, though self-limited and definite in its course, it is not strictly "specific," not contagious, and often not idiopathic but secondary.

We may admit that the presence of a special microphyte is nearly constant in the disease; so that its presence in the sputum, like that of Koch's vibrio in cholera-stools, is a useful means of diagnosis: nevertheless, the disease is a local inflammation; not a common inflammation produced by common irritants but an inflammation peculiar to the lung, and incapable of artificial reproduction. Of many, perhaps of most organs of the body we may say that they are capable of three kinds of inflammation at least: that which is acute and suppurative, marked by abundant exudation of leucocytes, by the presence of some forms of staphylococcus or streptococcus, and accompanied by what used to be called constitutional disturbance; secondly, a chronic interstitial inflammation with hardening of the tissue, shrinking and destruction of its peculiar elements by the new inflammatory growth; and, thirdly, an acute form of inflammation, not suppurative and peculiar to each organ. Pneumonia belongs to the last of these classes, and may be compared with acute Bright's disease, acute atrophy of the liver, and acute myelitis.

Of late years attention has been directed by Dr. Washbourn and others to the possibility of cases of acute pleurisy depending on the presence of the specific diplococcus of pneumonia, and running a short febrile course with the clinical features of pneumonia, but without the physical signs of hepatisation of the lung. If in such cases the lung really remains unaffected, and is found after death anatomically sound, while the pleura is the seat of a specific diplococcous inflammation, our notion of the disease pneumonia will be much modified; we shall have to admit it without reserve among the specific fevers, and to regard the inflammation of the lung as a very frequent but not a constant lesion.

Causation.—Of the causes of pneumonia we are still ignorant. Unless we disbelieve careful observations, because they do not fit a hypothesis, pneumonia can exist without the pneumococcus; and the same microbe may be found, not only in disease without pneumonia, but under healthy conditions also. Whether under certain circumstances this usually innocent microphyte acquires virulence, or whether a noxious and a harmless parasite resemble each other too closely to be distinguished, we cannot yet say; nor can we define the exact conditions which favour the occurrence of the disease. It is certain, however, that pneumonia is connected with cold, with north winds, with high ground, and with sudden fall of temperature.

Excluding bronchitis and ophthalmia, in which cold air seems to act as a direct common irritant, we may say that pneumonia affords the best evidence that there is truth in the common belief that a chill may "strike inward," and lends probability to the view that other acute inflammations—as pleurisy, colitis, acute Bright's disease, and even myelitis—may sometimes be due to a similar proximate cause.

We must also recognise as occasions of pneumonia certain previous morbid states, of which the most striking and important is influenza. Pneumonia following this disorder forms an important link between primary idiopathic pneumonia and the secondary pneumonia of fevers and septicæmia. Of the latter group of diseases, pneumonia appears to occur most frequently in typhus, less frequently in enteric fever, and in certain epidemics only of relapsing fever. It is not common in fatal cases of scarlatina or variola, and seldom takes the place of so-called lobular pneumonia in measles, whooping-cough, or diphtheria. In rheumatic fever its occurrence is happily infrequent. Among chronic diseases it is perhaps most frequent towards the termination of Bright's disease; but certainly it is not nearly so common as acute œdema of the lung, or pleurisy, or pericarditis. Pneumonia is probably more common, and is certainly not less fatal in the latter stages of diabetes. Beyond these there is perhaps no chronic disease in which its occurrence is sufficiently frequent to be of etiological or practical importance; but pneumonia is one of the intercurrent maladies which bring invalids to their end.

It is important to notice that lobar pneumonia is very rare as the consequence of phthisis, or of bronchitis, whether acute or chronic.

Natural history and incidence, distribution and local prevalence.—Pneumonia, as we see it in this country, is a sporadic and endemic disease. In its primary form it appears to be more common in winter and spring.

It is common all over temperate Europe, in the United States, and in the inhabited parts of the South Temperate Zone—in Australia and New Zealand, at Buenos Ayres, and in South Africa. It is less common in the Tropics; but on the hill stations of India it is far from infrequent during cold weather. It is also common in the highlands of Central Asia. In Cabul and Beloochistan it is ascribed, as in Italy, to the sudden change from the scorching heat of the day to the severe cold after sundown.

Pneumonia sometimes occurs in an epidemic form. From the Middle Ages downwards we have accounts of acute epidemic disorders, which seem more like pneumonia than anything else; and from time to time circumscribed epidemics are still reported in this country and in other parts of Europe. There is, however, no instance of prevalence of the disease so widespread as that of plague or cholera. All that happens is that a disease never uncommon becomes more common at certain times or in certain localities. This prevalence can sometimes be referred to a coincident prevalence of cold, dry winds, and sometimes is confined to a particular locality; but on the whole the phenomena do not seem to be of a different order from those which at certain seasons determine a greater prevalence of rheumatic fever, or quinsy, or bronchitis, or diarrhœa. What is more important is that we sometimes find a group of cases of pneumonia occurring together in the same village or in the same house. When influenza is epidemic, pneumonia follows it so often as to simulate an epidemic.

Sex and Age.—Pneumonia affects men more commonly than women—a preference usually explained by the greater exposure of men to changes of weather (Appendix, § 1).

No period of life is exempt from lobar pneumonia. It is, however, rare in infancy; and it is less common in children than in adults (Appendix, § 2).

It must be remembered that these facts concerning the incidence of pneumonia refer to the primary disease. Very little is known of the conditions under which pneumonia supervenes as a fatal complication of other diseases.

Prognosis.—There are few diseases of which the forecast varies so much with circumstances as it does in lobar pneumonia. Speaking broadly, primary pneumonia is much less dangerous than secondary; and in cases of primary pneumonia the danger increases, first, with the extent of the lung involved; secondly, with the age of the patient.

Age.—The latter point is one in which pneumonia agrees with most acute specific fevers—particularly with typhus, enterica, and variola. In this point of increasing danger with increasing age pneumonia is in striking and instructive contrast, not only with scarlet fever, but also with rheumatism and with diabetes.

Lobar pneumonia is rare in infancy and difficult to distinguish from extensive lobular catarrh; not only during life, but sometimes even after death. When present, however, as it often is in children between two or three and thirteen or fourteen years of age, lobar pneumonia is a most satisfactory disease to treat. Its symptoms and physical signs are well marked; the fever is high, and the condition of the patient threatening, but, with few exceptions, the temperature will fall on the fifth or sixth day, if not earlier, the symptoms will rapidly subside, and the recovery of the patient will be as safe and permanent as it is rapid.

With girls and boys between fifteen and twenty the extent of pneumonia is commonly greater than at a younger age; and the disease is more often severe and prolonged; but the prognosis is nearly or quite as good. Between twenty and five-and-thirty or forty the pneumonia of young adults is still of good augury on the whole; but at this time of life it is more frequent for both lungs to be affected. Intemperate habits begin to weigh in the seale; and cases of pneumonia, secondary to rheumatism, to influenza, to disease of the cardiac valves, to Bright's disease and to diabetes, begin to bring down the proportion of recoveries; although it is still high for idiopathic cases. After the age of forty acute pneumonia is always a serious disease. The prognosis in primary cases depends upon the temperate habits of the patient, on his being neither over-fed and obese on the one hand, nor, on the other, underfed and enfeebled by want and misery; on his being early put to bed under judicious treatment; on the extent of pulmonary tissue invaded, the height of the fever, the presence of cyanosis, and the effect of the pulmonary obstruction upon the heart. Secondary pneumonia at this age is a more dangerous disease than at the earlier period of adult life; but we see recovery from it in cases of enterica, of rheumatism, and even of Bright's disease and diabetes.

In old age pneumonia is a very fatal malady. As a secondary complication it frequently decides the termination of fevers, of chronic disease of the kidneys, of internal carcinoma, of lingering cases of hemiplegia, and other chronic affections of the brain and spinal cord. Even primary pneumonia, limited to a single lung, is dangerous in an aged patient, and we see recovery after the age of 70 with surprise. It does however occur, and sometimes even at so advanced an age as 80, but the cases are very rare.

Extent.—With respect to the area of pulmonary tissue affected, it is well known that double pneumonia is of graver prognosis than single (Appendix, pp. 133, 136). Pneumonia affecting the whole of one lung, from base to apex, though serious enough, is probably less so than that which affects a part of both bases. Apical pneumonia is more common in children than in adults, and probably for that reason is believed to be less dangerous. If, after one lung has partly or entirely cleared, pneumonia attacks the opposite one, the prospect of recovery is better than when both are hepatised at the same time.

Prognostic complications.—Of the conditions which affect the prognosis of pneumonia, probably the most important, apart from the age of the patient and the local extent of the disease, is intemperance. Such patients almost invariably become delirious, and the combination of delirium tremens and pneumonia is almost always fatal. Scarcely less serious is the presence of chronic renal disease; and next in gravity is that of valvular cardiac lesions. These two conditions, however, make the disease no longer primary but secondary.

Of the complications of idiopathic pneumonia, much bronchitis, and particularly bronchitis with bronchorrhea of the unaffected lung, is perhaps the most serious. Pleurisy, or even purulent effusion, is much less important; but pericarditis is a not infrequent and an extremely dangerous complication. Double pneumonia with pericarditis is almost invariably fatal (Appendix, p. 189).

A weak sound of the heart and a very rapid pulse are well-known indications of danger. Cyanosis, with very frequent breathing and action of the alæ nasi, is equally grave; and the two conditions often go together. They are partly due to the mechanical obstruction to the lesser circulation, and partly to the direct effort of the high temperature on the cardiac muscle; but in many cases both are aggravated by more or less pronounced sapræmia (24). Tympanites is a very unfavourable symptom in pneumonia, as in most other diseases; so also are hiccough and subsultus tendinum. When delirium persists during the day, and above all when it prevents sleep, the symptom is a grave one, and calls for decided treatment. Very high temperature indicates a severe case, and often goes with failure of the heart; but of itself hyperpyrexia is seldom fatal (Appendix, p. 140). Febrile albuminuria, even when abundant, is not of bad omen, and seldom or never persists after recovery. Free rusty expectoration or even hæmoptysis need give no anxiety. Diarrhœa is neither common nor dangerous, and if necessary it can be controlled without difficulty. Sweating during the height of the disease is not common, and when present is not of bad import. A profuse sweat, an abundant discharge of urine, or a sharp attack of diarrhœa, sometimes accompanies the critical fall of temperature.

Treatment. — *Historical sketch.*—Acute pneumonia is so striking and so severe a disease that as soon as it was definitely recognised it was attacked by all the resources of medicine. During the whole of the present century the treatment of pneumonia has reflected the various theories of disease and the changing practice of therapeutics.

The conception of pneumonia current at the end of the last century was that of an acute inflammation, directly produced by cold, and attacking a healthy subject. The business of the physician was to combat the enemy by the potent weapons of bleeding, blistering and starvation, aided by purgative and alterative drugs. The high fever, the flushed face, the acute pain and the burning skin were evidence of a "sthenic" inflammation. The physician felt confident that by antiphlogistic remedies he could subdue the disease; and his only fear was lest the patient's strength should fail under the necessary treatment, that he might die, not of the disease, but of the weakness attending its cure—*mort guéri*. The "corroborant" practice of the Brunonian school of medicine never obtained such vogue in England as on the Continent. It was as baseless as the iatro-chemical or the iatro-mechanical systems which prevailed earlier in the 18th century, and had deservedly fallen into disrepute. During the first half of the present century the antiphlogistic treatment of pneumonia and of other acute inflammations continued to be the only one followed in civilised countries—in Dublin as well as in Edinburgh, in Vienna as well as in Madrid; and precisely the same treatment was adopted by surgeons for compound fractures, inflammations of the eye, and for what we now call pyæmia. To realise the confidence and energy with which this absurdly called "heroic" treatment was carried out, one must have seen, as I saw so late as 1863-64, the treatment not only of pneumonia and pericarditis, but of rheumatic and typhoid fever, by Bouillaud at the Charité; or one must read the lectures of the late Dr. Peter Latham (1845), in which with admirable rhetorical skill he enforces the dogmas of the day.

The only important modification of the antiphlogistic treatment of pneumonia introduced during the period between 1790 and 1850 was the introduction of large and repeated doses of antimony by Rasori (1808), a practice much followed for a time both in Italy and France. The undoubted effects of this drug, in producing nausea and disinclination to food, lowering the blood-pressure, and causing diaphoresis, were quite in harmony with the effects of bleeding, purging and salivation.

It was and still is true, when a patient is suffering from acute pain in the side with fever and a frequent, strong, and hard pulse, that venesection and free purging will relieve the pain, reduce the arterial tension, and give him grateful relief from his sense of fulness and oppression. It was no doubt from observation of these effects, which were well known to Sydenham, Mead, and Boerhaave, that the antiphlogistic practice began; and when the discoveries of Laennec made it possible to recognise pneumonia from the first, and to trace its daily progress, it seemed right to continue and to reinforce the treatment apparently so appropriate.

The mistake lay in having no control-observations. Physicians saw patients in an illness apparently desperate, and under treatment by bleeding and antimony they saw most of the symptoms relieved; frequently, after a battle of several days, the disease was subdued and the patient convalescent; but they did not know, because they never ventured to try, what would happen if these remedies had been omitted. It is a humiliating but instructive fact that the possibility of recovery from acute disease without active treatment was established by the assumed success of a demonstrably futile system of therapeutics, the last, we may hope, of attempts to answer the absurd question, "On what universal principle should disease be treated?" When it could not be denied that persons suffering from pneumonia and other acute disorders did recover when treated with infinitesimal doses of useless drugs, it could not be long doubted that some acute diseases might get well of themselves.

The report of some cases of pneumonia which recovered in the Homœopathic Hospital at Vienna awakened thought on this subject, and an article by the late Sir John Forbes, which appeared in the *British and Foreign Medical Chirurgical Review* (1846), pressed the lesson home. Skoda had given fair trial to other methods of treatment, and found that his mortality from acute pneumonia was much less than when treated by bleeding, blisters, and antimony. These facts were made known in England by Dr. Geo. Balfour, who had followed Skoda's practice in Vienna (6). Dr. Hughes Bennett of Edinburgh also published a series of cases of pneumonia treated without bleeding, antimony, or mercury with unusually small mortality (1848); and he gave an interesting account of the arguments of Alison, Watson, Christison and Markham (8). Discussion followed, but it was less prolonged than might have been supposed; as so often happens, general opinion had been gradually altering, and was ready to turn at the first summons. Moreover, the advocates of antiphlogistic treatment threw away their case by the assertion that they were right in bleeding before, and right in doing nothing afterwards—not because their opinions but the nature of the disease had changed; and a presumed "sthenic type" of fevers and inflammations, with a successful heroic treatment corresponding thereto, was dwelt upon with the same satisfaction that an old man contrasts the hard frosts and heroic exploits of his youth with the mild winters and feeble powers of his contemporaries. For a long time the antiphlogistic treatment held its ground in books and lectures; but those who taught it always found in practice an excuse for disobeying their own precepts. By 1860, however, the change in treatment was nearly universal; and during the latter half of the 19th century, English physicians, under the guidance of Jenner and of Gull, have given up the "heroic" treatment of pneumonia.

In too many cases the treatment which supplanted it was of a purely negative kind, disguised under such platitudes as the prescription of rest in bed for a patient who could neither sit up nor rest; of light and nourishing food, as if the opposite was ever ordered for a fevered man; and of avoiding cold for a patient with a temperature of 104°.

At present we may hope that a more rational system is established. We know that under favourable circumstances pneumonia needs no treatment beyond the following of the indications of the patient's own feelings, and awaiting the favourable result which will follow in the course of a week.

Abortive treatment.—It is clear that no means known at present can cut short pneumonia. There is nothing absurd, however, in supposing that this may one day be done. We do cut short the manifestations of syphilis and of ague, of hydrophobia, and of diphtheria.

Since the presence of microbes has been ascertained in pneumonia, and the pathogenetic significance of the diplococcus of Talamon and Fränkel has been admitted by physicians with more or less confidence, it is not surprising that attempts have been made to apply the theory of immunity in treatment. The method adopted has been to render animals immune from the disease by introduction of the supposed pathogenetic microbe in doses of increasing strength until this immunity is attained, and then to inject the serum of such an animal into the veins or tissues of patients suffering from pneumonia. This practice was introduced by F. and G. Klemperer in 1891, and has been followed by some apparent success (21).

Whether pneumonia can be cut short or not, it may be successfully guided. No reasonable observer would deny that, although we are rarely able to say that a patient's life was saved by such and such a timely measure, yet in the long run the expectant method of treatment, which interferes only as occasion requires, is followed by a far lower mortality than misplaced attempts to "jugulate" the disease, or than a completely negative treatment.

General treatment of uncomplicated cases.—In a case of primary pneumonia in a young subject our first care should be to keep him cool by light covering, cradles under the bedclothes, and frequent sponging with cold or tepid water. He has no appetite, and there is no necessity to force food upon an unwilling and often flatulent stomach. If the patient will drink two pints of milk, or one pint of milk and one of broth, in the twenty-four hours, he will not starve. Stimulants should not be given until required by some special indication.

The thirst, the parched tongue, the fever, the scanty and concentrated urine, and the hot, dry skin all call for drink; and the patient should be allowed to take as much cold water as he pleases. It relieves his thirst, it moderates the sensation of heat, it flushes the kidneys, by inducing perspiration it relieves discomfort, and by evaporation it helps to lower the temperature. If patients prefer effervescing drinks they may have them, and milk with soda water is often the pleasantest mode of supplying nourishment; but it must be remembered that milk is food, and to keep a pneumonic patient on milk and beef-tea without water is a practice as unphysiological as it is disagreeable. The exception is when a child refuses nourishment and will only drink milk when compelled by thirst. Toast and water, barley-water with or without a slice of lemon, tamarind or red-currant water may be given according to the patient's preference; and, although the cold and unadulterated element is as a rule most grateful, some patients, particularly if troubled by gastric disturbance, much prefer to drink hot water. There is no reason for withholding tea as a beverage; but this and other indulgences the patient will probably enjoy the more if not given until asked for. Oranges or grapes are more pleasant in convalescence than during the height of the disease; but there is no objection to their use at any time.

With regard to drugs, though an uncomplicated case of pneumonia will do well without them, yet long and wide experience shows that solutions of neutral salts are of service in diminishing the sense of heat and tension, and in promoting secretion. Nitre is perhaps the best of these; but citrate or acetate of potash or acetate of ammonia act in a similar way; the potash salts are supposed to act most on the kidneys, and those of ammonia on the skin. They may be given with chloroform or peppermint, or in any bitter infusion such as serpentary, orange, or quassia. They are not necessary, but beside their undoubted, though slight, physiological effects, an occasional draught of medicine is liked by most patients, and it helps to keep up the attention of the nurse.

It is an old custom, and perhaps a wise one, to administer a purge at the beginning of any acute disease. The furred tongue, the headache, and the customary constipation seem to call for it; and it helps to prevent flatulence and so to favour respiration in the course of pneumonia. A blue pill, followed by a black draught—or, what is much pleasanter and nearly as efficient, by sulphate of magnesia with bicarbonate of soda in a carminative vehicle, or some other natural or artificial solution of purgative salts, are the best ways of meeting this indication. An efficient action of the bowels on the first day of the disease has the important advantage of setting the mind of the patient and of his nurses at rest, and of preventing unseasonable purging afterwards.

From the first day constant attention should be directed to the chart of temperature, to the pulse and respirations, and to the auscultatory signs. The chest should be thoroughly examined on the first or at least on the second day; and, when the diagnosis and seat of the disease are thus made clear, the frequency and minuteness of subsequent examinations may depend upon the patient's condition. It is often wise to refrain from rousing a patient from sleep for this purpose, or from exciting the resistance of a child. In such cases, or when delirium or extreme weakness forbids a thorough physical examination, we may generally judge of the condition of the lungs by counting the number of respirations, by noticing the colour of the face and lips, and by observing the action of the nostrils and muscles of forced respiration. By gently rolling the patient on his side, first one and then the other, we can obtain sufficient evidence of the state of the lung, by means of the flexible stethoscope, without raising him in bed.

Treatment of special symptoms.—We now come to the treatment of symptoms, ordinary or extraordinary.

The temperature is always or almost always high, and hyperpyrexia is frequently met with; but it is less common than in rheumatism, typhus, or scarlatina, and its danger is certainly less than in rheumatism. A temperature of 104° demands attention, but in children and young adults does not in itself require interference. When it rises above this point a tepid bath for a child, and sponging the body with cold water in an adult are indicated. In some cases it is more convenient to put bladders or india-rubber bags filled with ice in a man's armpits and between his thighs. If the temperature rise above 105°, frequent sponging with ice-cold water, rubbing with ice, or immersion in a bath at a temperature of 80° is called for.

In some cases wrapping the patient in a sheet wrung out of cold water is more convenient or less unpleasant, and it is an efficient means of relieving moderate pyrexia; or Leiter's tubes may be carried in coils round the head and placed over the great blood-vessels, above the collar-bones, in the armpits, and in Scarpa's triangle.

In children a high temperature is more frequent and less injurious than in adults; whereas in elderly patients a temperature above 103° is a serious matter, and cooling measures must be sedulously and yet cautiously employed.

Whatever the temperature which appears to call for interference (and in this we must be guided not only by the thermometer, but also by the mental condition and the state of the pulse), direct application of cold should be the treatment adopted. Antipyretic drugs are either inefficient or their action is transient; and they often cause dangerous depression of the heart's action or complete collapse. Salicyl-compounds are only indicated when pneumonia occurs as a complication of rheumatism; and full doses of quinine only in the case of persons who have suffered from malaria. Antipyrin or phenacetin is sometimes useful for relieving severe headache; but even then they must be used cautiously. When headache is troublesome, the application of ice to the head often gives the greatest relief. A dose of bromide is sometimes efficient, or the aromatic spirits of ammonia, or a strong cup of coffee.

The pain of the pleurisy which always accompanies pneumonia varies greatly in its severity and duration. Often it is so slight that a warm poultice, or an ice-bag, or rubbing with menthol is enough to allay it. In severer cases two or three leeches are sometimes of striking benefit; or, if there be no counter-indications, the sixth or the fourth of a grain of morphia may be injected under the skin. When the pain is persistent, a blister relieves it more certainly than any other remedy.

The effusion of pneumonic pleurisy is seldom large enough to demand special treatment. If it should be considerable, it is best to aspirate at once; if it be small, it will often disappear of itself after the crisis, or may be dealt with during convalescence by blisters and diuretics or, if these fail, by paracentesis.

Empyema is the most important sequel of pneumonia. When its presence is discovered, it should be tapped at once, and afterwards incised and drained. It is said that if the diplococcus of pneumonia be present alone in the exudation, the pleura will recover without fresh secretion of pus; whereas if colonies of staphylococcus or streptococcus are found, it is better not to aspirate but to incise at once, or the pus is sure to form again: that the latter rule is true, at least for adults, is supported by general experience; the former statement is more disputable.

One of the most formidable complications of pneumonia is pericarditis. It is most common in cases of double pneumonia with well-marked pleurisy, and may be chiefly fibrinous, or accompanied with large effusion of serum, or occasionally purulent. Its recognition is often far from easy. Marked orthopnœa, an irregular or intermittent pulse, and precordial distress are each of them valuable signs of pericarditis, but they are neither constant nor infallible. In every case of pneumonia the apex and base of the heart should be examined each day: from the former to learn the strength and character of the first sound of the heart, from the latter to detect the earliest sign of pericardial friction. This is difficult to make out when noisy and frequent breathing obscures the cardiac sounds; and the difficulty is often increased by the restlessness and distress of the patient. When it is impossible to obtain even a momentary pause in breathing, close attention to the pulse will impress the cardiac rhythm on the ear; and when this is once done, the practised auscultator can neglect the bronchial râles almost as much as the noises going on in a room. At least we may say whether the sounds are normal or accompanied by a murmur; and in the latter case, if we know that they were normal on the first day of the illness, the murmur is most likely a pericardial rub.

It is sometimes impossible to detect a large pericardial effusion by percussion. We must depend upon the situation and force of the cardiac impulse, on the faintness of the cardiac sounds, and on the signs of downward pressure of the left lobe of the liver. If called for the first time to a case of acute pneumonia with pericarditis we may find the diagnosis extremely difficult.

Our treatment of pericarditis, when recognised, is unfortunately not yet very efficient. There is no reason to believe that bleeding, leeching, or mercury has any effect on the inflammation. A blister, however, over the cardiac region will often relieve the precordial oppression; and twice I have seen it signally and demonstrably successful in removing the signs of friction and of effusion. In a severe case of pneumonia, however, blisters are undesirable; and if we believe that pericardial effusion is extensive it is probably better practice to introduce a hypodermic syringe at once and draw off a few drops of the fluid: if serous, the pericardium may then be aspirated; if purulent, it should be incised and drained like an empyema. I have repeatedly tapped the pericardium, and have never seen harm to follow the practice; on the other hand, I have been unfortunate in not witnessing the marked relief which many physicians have recorded. Several most successful cases of draining the pyo-pericardium have been published.

The most imminent danger in pneumonia is dyspnœa from extensive consolidation of the lung, overloading of the right side of the heart, and arterial anæmia. We are at present powerless, or nearly so, to limit the spread of hepatisation; all we can do is to help the patient to outlive the stress of the disease.

For this purpose frequent feeding with small quantities of nutriment is necessary. The most useful forms of nourishment are milk, raw eggs, beef-tea, and meat jelly, or one of the various meat extracts in the market. Food should not be given oftener than every two hours. When there is vomiting, or when the patient refuses food—as sometimes occurs in a child or in an adult who is delirious—it is best to abstain altogether from feeding by the mouth, and to use instead a nutriment enema or nutrient suppositories, after the rectum has been washed out.

In cases of secondary pneumonia, and in primary cases occurring in later life with few exceptions, alcohol is indicated, and in all cases when the pulse is irregular or very rapid, and the first sound of the heart weak. It may be given in the egg-and-brandy mixture of the Pharmacopœia, or diluted with water as a beverage. Its effect should be watched, and the amount and frequency of its administration regulated by its effects. It often improves the pulse and soothes the brain; when these effects are apparent, it should be pushed freely. Half an ounce given every four hours is suitable for an uncomplicated case of pneumonia in a patient over fifty years of age. Six or eight ounces in the twenty-four hours are needed in severe cases with feeble circulation; and as much as twelve ounces when by the patient's symptoms he seems to demand it, and his state to improve under the remedy. In some cases champagne is better tolerated than brandy, whisky, or rum, and has as good or perhaps a better effect. Sometimes, however, we find that any form of alcohol causes excitement and discomfort without strengthening the pulse; in such cases it is best to omit it for a time, and to rely upon strong beef-tea and strychnine.

As the pulse affords the chief indication for the administration of brandy, so the state of the patient's breathing guides our use of ammonia. This admirable drug acts not only on the heart, but also on the respiratory centre, stimulating the reflex action of coughing, and so clearing the air-passages. Like other alkalies, its action on the bronchial secretion is to make it thinner and more easily got rid of. In all cases of pneumonia, except those affecting a single lung in children or healthy young adults, carbonate of ammonia should be given in four or five-grain doses combined with syrup of tolu, liquorice, or treacle, to soften its pungency. Compound tincture of cardamoms or lavender may be added with advantage; or the drug may be given dissolved in peppermint or chloroform water. It should be repeated every four hours or more frequently, up to five grains every hour, if dyspnœa and cyanosis threaten imminent danger.

There is always some bronchitis with pneumonia, and this may add considerably to the dyspnœa; but bronchitis is most serious when in a case of unilateral or extensive pneumonia it affects the healthy lung or the healthy part of one. It is in such cases, perhaps, that carbonate of ammonia is most signally useful.

In addition to brandy and ammonia, or in cases where one or the other seems to fail, we may use strychnine as an efficient stimulus of the centres in the bulb. It is most valuable in cases of failure of the heart's action, when alcohol seems useless or even mischievous to the patient; five drops of liquor strychninæ may then be given with excellent effect, and repeated should occasion arise. Strychnine is much more useful given in one or two full doses than in smaller ones frequently administered. Of course it must never be given with ammonia. When a pneumonic patient is at the point of death, three or four drops of solution of strychnine injected under the skin of the arm are more effectual, and less liable to lead to local abscess than brandy, ammonia, or ether administered in the same way; but each of these drugs thus exhibited has undoubtedly saved life.

In severe cases of pneumonia, the rapidity of the pulse, its frequent irregularity, and the low blood-pressure naturally suggest the use of digitalis. It is generally prescribed along with other remedies, and it is therefore difficult to estimate its individual effect; but my own experience has been disappointing, and my disappointment is shared by many physicians who have used it since Traube recommended it fifty years ago.

In pneumonia its effect is incomparably inferior to that which we see every day in mitral regurgitation, with dropsy and rapid, weak, irregular pulse. The experiments of Drs. Brunton and Cash (8) indicate that the effect of digitalis on the heart is greatly weakened by pyrexia. It has been conjectured that the right ventricle, the cavity most burdened in pulmonary obstruction, has not muscle enough to utilise the drug; but, if the objection were theoretically admissible, it is contradicted by the fact that it is this right ventricle which we successfully stimulate and control in cases of mitral regurgitation. Whatever the explanation, digitalis would probably be seldom employed if our only experience of it were in pneumonia. At the same time, it is right to add that some good observers, both at home and abroad, have a much more favourable experience of this powerful drug in the disease. Dr. Petrescu of Bucharest reports a remarkable low percentage of death in pneumonia treated with large doses of the powdered leaves or of the infusion of digitalis.

An ancient stimulant, which had long fallen into disuse, has been lately revived in cases of pneumonia, enteric fever, and other exhausting diseases; namely, musk, given in ten-grain doses, and repeated in three or four hours. It is very expensive, and often it fails entirely; but I have seen it produce striking improvement for a time in severe cases of bronchitis, pneumonia, and fever; and in four or five of these instances it probably saved the patient's life.

When dyspnœa is urgent, and the patient apparently dying of cyanosis, the inhalation of oxygen is a rational mode of treatment, and has been advocated for many years past. It is now possible to obtain the gas ready made in large iron cylinders much more conveniently and cheaply than before; and it sometimes proves remarkably useful. It seems never to do harm, and it is a matter of surprise that its effects are not more uniformly and obviously beneficial. It is possible that we have yet to learn how to use it most efficiently. At present it takes its place among the adjuvants of successful treatment.

Delirium is an important complication of acute pneumonia; this is often best treated by an extra dose of brandy in the evening. In the case of an intemperate patient digitalis is here a valuable drug. Bromide of potassium, or full doses of henbane or chloralamide, may be used with good effect. When coincident with high temperature, a wet pack or cold sponging is often the best cure for delirium.

Sleeplessness is a frequent and trying symptom. In some degree it is inevitable, and, when there is much bronchitis, to prolong sleep might be dangerous to the patient; but in other cases the insomnia is purely injurious, and must be met by every means in our power. In ordinary cases a cup of beef-tea with a spoonful of brandy is an excellent sedative; and if, before this is taken, the patient has been well sponged, furnished with a clean night-dress and a fresh pillow, if the head is kept high and as cool as possible, and the room dark and quiet, natural sleep will often follow. Nothing is worse at such times than meddlesome nursing, moving about on tip-toe, conversing in whispers, and smoothing the patient's pillow. The administration of food or medicine may well be omitted for three hours, or even longer. In cases where this would be dangerous the patient is not likely to sleep too long.

The refreshment of sleep at some time of the day or night is of primary importance in pneumonia as in other fevers; and when the means above mentioned fail, and the patient has been sleepless for twenty-four hours, the question arises of exhibiting our most powerful remedy, opium. It has been forbidden lest its use should increase cyanosis, diminish respiratory efforts, and lead to fatal coma. These fears are far from groundless. When there is extensive consolidation or much bronchitis, when the patient is becoming livid, and the expectoration scanty, it would be bad practice to give this drug. Mustard plasters, strychnine subcutaneously, and ammonia by the mouth are the remedies indicated. Often during a whole night the patient must be restless, must continue to cough and expectorate, and must use every muscle available to keep himself alive by forced breathing. The orthopnœa, the coughing, the sleeplessness, the distress are all evidence of the struggle for life; and the worst sign is when a cyanotic patient lies low in bed, drowsy, with weak and shallow breathing, the respiratory centre in his bulb poisoned by carbonic acid. After such a restless night as we have just described the patient will often find the breathing relieved when morning comes; and, after taking a cup of coffee or a glass of wine, he may sink into natural slumber, and awake refreshed and ready to renew the struggle.

But in many cases of pneumonia the danger is not directly from suffocation; it is rather from the effects of a continued high temperature upon the heart, the impending weakness of the respiratory muscles, and exhaustion of the reflex activity of the nervous centres. In such cases 10 grains of Dover's powder, 5 grains of a compound soap pill, 15 or 20 drops of laudanum are often invaluable, and succeed when all other hypnotics are powerless. An additional warrant for the use of opium is dilatation of the pupils. The presence of albumin, if only of pyrexial origin, is no counter-indication; but if the patient be a subject of chronic Bright's disease we must forgo the use of this valuable drug, or use it at his peril, to escape a still more imminent danger.

There are two remedies which have fallen into general disuse, but each of them worthy of being employed on occasion.

One is the use of emetics—antimonial wine, or ipecacuan in full doses, or sulphate of zinc, or subcutaneous injection of apomorphia, or a draught of mustard and water and tickling the fauces with a feather: such drugs, in emptying the stomach, also get rid of accumulated bronchial secretion, and produce deep and efficient respiratory effort. This method of treatment is still much used with children suffering from broncho-pneumonia, for in them vomiting is easy and expectoration difficult. In adults a vomit and a purge no longer form a routine prescription; and, although no doubt an emetic is sometimes useful, it is an unpopular remedy, and its effects are often disappointing. Not infrequently even large doses of the emetic fail to excite vomiting, and the patient's condition is then uncomfortable to himself and embarrassing to the physician. A full dose should always be given, and in pneumonia and bronchitis stimulant emetics like mustard and sulphate of zinc are more suitable than antimony. Though often disappointed in this plan of treatment, I have seen cases in which it was of undoubted benefit.

The other ancient remedy is that of bleeding. We saw that it was once employed to subdue fever, and to cut short inflammation, and that its use for these purposes is deservedly discredited. Venesection is less potent for good and also for evil than used to be thought, but it is not to be forgotten or neglected. Its effect in relieving the pain of aneurysm was insisted on by the late Dr. Hughes Bennett in the midst of his attack upon its use in pneumonia. The same iconoclastic reformer also recognised its value when used in the very first stage of pneumonia before dulness had appeared. It is not often that a patient is seen in this stage, which is usually very short; but when pneumonia occurs as a primary attack in a young and robust subject, with severe pleuritic pain, I would advise bleeding, not as a cure, but as a means of relief. If the pulse be full, strong and hard, and a great sense of præcordial oppression be present, the withdrawal of 6 or 8 ounces of blood from the arm, by temporary lowering of the arterial pressure, will sometimes remove distress better than any other remedy, and will leave, if not a beneficial, at least no deleterious effect on the subsequent course of the disease.

In cases of cyanosis, with a small and feeble pulse, congestion of the surface, and distension of the right ventricle, as shown by epigastric pulsation and pulsation of the great veins, our object is not to lower the arterial blood-pressure, but to relieve the over-pressure in the right side of the heart and the systemic veins. The withdrawal of 10 or 12 ounces of blood under such circumstances is a rational procedure, and in practice is often successful in tiding over a dangerous period of the disease. In my experience, however, venesection in the cyanosis of pneumonia is less strikingly useful than in the corresponding phase of bronchitis; although it is more so than in the cyanosis of valvular disease of the heart.[1]

In the treatment of convalescence from pneumonia we have fortunately little to discuss. Like typhus, and in striking contrast to enteric fever and scarlatina, acute lobar pneumonia is a disease which either kills the patient or leaves him much as he was before. When the crisis is past the inflamed lung very seldom fails to clear up rapidly and completely.

As soon as the temperature falls, brandy and medicine should be omitted or greatly reduced in amount and in frequency of administration. Sleep should be encouraged, and food given in accordance with the patient's returning appetite. Wine is often useful during the first days of convalescence; or, if the patient prefer it, malt liquor may be taken with at least equal advantage. There is no danger of catching cold, and the patient may go out of doors in favourable weather as soon as he desires to do so.

Appendix of Cases

The statistics subjoined refer to 434 cases of (acute fibrinous) pneumonia collected from the following sources: 329 schedules were filled up from the records of Guy's Hospital during years 1891-94 by the medical registrar, Dr. J. H. Bryant, assisted by Mr. F. J. Steward, and to both these gentlemen I am greatly indebted for this valuable help. I have added 32 hospital cases of my own, admitted in 1895 and 1896; the remaining 73 were private cases seen in consultation. The number of cases is not large enough for all purposes, but it is, I hope, large enough to be of service for others.

The facts tabulated in the schedules were:—(i.) the sex, and (ii.) the age of the patient; (iii.) the assigned cause, the antecedents, and initial symptoms of the attack; (iv.) the part of the lungs affected; (v.) some of the chief symptoms, particularly the highest temperature attained, the presence and characters of the sputa, the presence of herpes, and (vi.) any important complications or sequels; (vii.) the duration of the disease, reckoned from the rise of temperature to the end of pyrexia, and, lastly, (viii.) the result in recovery or death, with details of the fatal cases.

1. **Sex.**—Of the 434 cases, 320 occurred in male, and 114 in female patients, a somewhat larger disproportion than usual. The difference is most marked in early adult life, least in children, and disappears in the statistics of prisons, where both sexes are under similar external conditions, and where epidemic pneumonia would affect both alike.

2. **Age.**—The following is the incidence of the 434 cases at the several periods of life.

(*a*) *Under five years.*—Total, 29 patients. Of these, 6 were above four years old; 9 were between three and four; 10 were between two and three; 1 was eighteen months old; and 2 were under a year—one four and the other seven months old.

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[1] See forty-nine cases of venesection in the *Medico-Chirurgical Transactions* for 1891.

Between five and ten years there were 66 cases, making a total of 95 patients under ten years old.

Between ten and fifteen there were 45 patients, and *between fifteen and twenty* 48, making a total of 93 between ten and twenty.

(*b*) Arranging the figures in perhaps a more instructive way, we have 3 cases in infancy, 26 in early childhood (two to five), and 159 between five years old and adult age.

Between twenty and forty there were 149 cases.

Between forty and sixty there were 74 cases.

Between sixty and seventy there were 18 cases; showing a greater frequency than between fifty and sixty, if we allow for the fewer possible patients at the more advanced age.

Above the age of seventy there were 5 patients—2 aged seventy-two, 1 seventy-three, and 2 seventy-five.

These numbers confirm what is a matter of general observation, that lobar pneumonia is rare in infancy, very common between two years old and twenty, gradually less common in adult life, more rare after fifty. The cases in later life are mostly secondary.

(*c*) Arranged in decades the numbers are—

Between 3 months and 10 years	95	Between 40 and 50 years .	. 54
,, 10 and 20 years .	. 93	,, 50 ,, 60 ,, .	. 20
,, 20 ,, 30 ,, .	. 87	,, 60 ,, 70 ,, .	. 18
,, 30 ,, 40 ,, .	. 62	,, 70 ,, 75 ,, .	. 5

3. Origin and onset.—In 38 cases only was the attack of pneumonia explicitly attributed to a shortly precedent exposure to cold; a "chill" received more than a week before the illness began was not counted. In 14 cases the origin of the disease was imputed to an injury or "accident"; but I have never seen a case in which this supposition was borne out.

In 3 cases only was there a probability of *infection* from another case in the same house. Two of these patients were children; the third was a lady who was attacked with pneumonia while nursing her child with the same disease and while her husband was convalescent from it. Another patient, whom I saw with Dr. Charles Addison at Colchester, was one of no less than four cases of pneumonia in the same house; but in each of them the pneumonia had been preceded by influenza, so that the cases should come under another heading.

In the hospital cases it is likely that many more had their origin in *influenza;* but in only three was the sequence definitely recorded. Of 73 private cases, influenza had preceded the pneumonia in 8.

The *onset* of the disease was gradual in not less than 93 cases, an unexpectedly large number—more than a fourth of the whole. In the remaining 234, in which the early symptoms were clearly ascertained, the onset was sudden; it began with well-marked shivering in 95 cases, with vomiting in 50 (chiefly children), with convulsions in only one case (a child), in the remainder with sharp pain in the side, or once or twice with syncope.

A previous attack had occurred in 18 cases—in 3 within a year, in 3 within two years, in 1 three years ago, and in 3 so long as fourteen, seventeen, and eighteen years before the second attack. One patient had suffered five or six times from the disease, two had a fourth, and one a third attack.

4. Locality.—The right lung was, as generally observed, more often affected than the left; but the difference was due to the large excess of right-sided over left-sided apical pneumonia. When the base only was affected, there were in my tables rather more cases on the left than on the right side.

Cases beginning in the right base	140	291 basal.
,, ,, left ,,	151	
,, ,, right apex	60	80 apical
,, ,, left ,,	20	
,, affecting the middle part of the lungs—6 right, 2 left		8
,, ,, both lungs		55
		434

Total number of cases affecting the right lung only	206
,, ,, ,, left ,,	173
,, ,, ,, both lungs	55
	434

Double Pneumonia.—The 55 cases in which both lungs were affected seem to deserve separate attention, particularly in their relation to age and to prognosis. The table explains itself.

Sex.	Age.	Locality.	Complicated by	Complicating	Result.
M.	31	Both bases	Tympanites	...	Died
M.	43	R. base, L. apex	...	Chronic phthisis	Died
M.	49	Both bases	...	Chronic phthisis	Died
F.	12	Both bases	Gangrene	Spinal caries	Died
F.	4	Both bases	...	Chronic otorrhœa	Recovered
M.	36	Both bases	...	...	Recovered
M.	49	Both lungs	...	Morbus Brightii	Died
F.	46	Both bases	Gangrene, Endocarditis	...	Died
M.	6	Both bases	Empyema	...	Recovered
M.	36	Both lungs	Gangrene	Intemperance	Died
M.	21	R. base, L. apex	Diarrhœa	...	Died
M.	15	Both lungs	...	...	Recovered
M.	39	Both bases	...	...	Recovered
F.	18	Both lungs	Laryngitis	...	Recovered
M.	14	Both lungs	...	...	Recovered
M.	22	Both lungs	Empyema	...	Died
M.	39	Both lungs	...	Admitted moribund	Died
M.	8	Both lungs	Empyema, Pericarditis	...	Died
M.	17	Both lungs	...	Admitted comatose	Died

Sex.	Age.	Locality.	Complicated by	Complicating	Result.
M.	32	Both lungs . .	...	Intemperance . .	Recovered
M.	48	Both lungs . .	...	Intemperance . .	Died
F.	18	Both lungs .. .	...	...	Died
M.	4	Both lungs . .	...	...	Died
F.	3½	R. base, L. apex .	...	...	Died
M.	28	Both bases . .	...	...	Recovered
F.	21	Both lungs . .	...	Puerperium . . .	Died
M.	30	Both lungs . .	...	...	Recovered
M.	23	Both bases . .	Pyopneumothorax	Intemperance . .	Died[1]
F.	13	Both apices . .	...	...	Recovered
M.	45	Both lungs . .	...	...	Died
M.	37	R. base, L. apex .	Delirium tremens	Cirrhosis of liver . .	Died
M.	52	Both lungs . .	...	Intemperance . .	Died
F.	28	Both lungs . .	Empyema . .	...	Recovered
M.	20	Both lungs . .	...	...	Died
M.	19	Both lungs . .	...	...	Recovered
M.	30	Both lungs . .	...	...	Recovered
M.	6	Both lungs . .	Empyema . .	...	Recovered
M.	7	Both lungs . .	Empyema . .	Chronic otorrhœa . .	Recovered
M.	18	Both lungs . .	Empyema . .	Influenza . . .	Recovered
M.	12	Both apices . .	...	...	Recovered
M.	33	Both lungs . .	Laryngitis . .	...	Died
M.	22	Both lungs . .	Laryngitis . .	...	Died
F.	27	Both lungs . .	...	...	Recovered
M.	20	R. and then L. base.	...	...	Recovered
F.	48	Both bases . .	...	...	Died
M.	35	Both bases . .	...	Intemperance . .	Died
M.	54	Both bases . .	Endocarditis, Meningitis	...	Died
M.	17	L. base, then R. apex	...	...	Recovered
M.	35	L. base, R. apex .	...	...	Died
M.	48	Both bases . .	...	...	Recovered
F.	43	Both bases . .	...	...	Died
M.	20	Both bases . .	...	Intemperance . .	Recovered
M.	9	R. base, L. apex .	...	...	Recovered
M.	12	Both bases . .	...	...	Recovered
M.	20	Both bases . .	...	Mania	Died

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[1] He left the hospital against advice.

Summary.—Deaths, 30; recoveries, 25. Only one recovery took place among patients who were over forty years of age when attacked. This was in the case of a man aged forty-eight with pneumonia of both bases.

5. Symptoms.—Those which I particularly recorded were the temperature, the character of the sputa, and the presence of herpes labialis.

Albuminuria was frequently reported, and in cases when stated to be absent an earlier or more frequent examination might often have found it. Its presence from more or less latent Bright's disease, from acute nephritis or renal embolism, and the occurrence of traces of albumin from leucorrhœa, cystitis, spermatorrhœa, or gonorrhœa virulenta would also disturb the results.

Temperature.—In two cases this is recorded as subnormal; both these patients were admitted into hospital in a collapsed and moribund condition, and died soon after being got to bed. In the rest the highest point observed was as follows:—

Degrees Fahr.	Cases.
100–100·4	5
101–101·8	22
102–102·8	55
103–103·8	114
104–104·8	164
105–105·8	42[1]
106 (*bis*), 106·4, 106·6	4[2]
107·8	1[3]
109	1[3]
	408

Herpes.—A herpetic eruption was noted in only 53 patients. Of these, 46 recovered and 7 died.

Sputum.—Notice of this is not always explicitly made, and patients may have died too soon for it to be seen. Of the 290 cases in which definite statements were made, there were—

116	in which the sputum was "rusty."
7	„ „ „ "greenish."
24	„ „ „ mucous or muco-purulent.
13	„ there was free hæmoptysis.
130	„ no sputum was expectorated.

In 113 cases of children under fifteen expectoration was absent. The only children who succeeded in coughing up their sputa were 4, all between eleven and fifteen years of age, who brought up rusty sputa; and 3 between six and eight years old, who coughed up blood-stained mucus.

Of adults who did not expectorate at all during the whole attack of pneumonia there were as many as 17. Three of these were between fifteen and forty (out of 194); 5 were between forty and fifty (out of 52); 3 between fifty and sixty (out of 16); 3 between sixty and seventy (out of 20); and 3 between seventy-two and seventy-five (out of 5).

6. Complications and sequels.—In five patients the pneumonia ended in *gangrene* of the lung; one a case of chronic caries of the spine, one complicated with pericarditis, one with ulcerative endocarditis, and one with delirium tremens. All of these five patients died.

Ulcerative, septic *endocarditis* occurred six times, and in three of these *meningitis* was also present; while in two other cases meningitis was found after death. *Pericarditis* was a complication in fourteen cases, and in one of these there was also endocarditis (in addition to the six above mentioned).

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[1] Of these forty-two, only four reached 105·8°, and nearly half did not exceed (so far as was noted) 105°.

[2] Two of these patients recovered after temperatures of 106° and 106·6°.

[3] Both of these patients died.

Icterus occurred in four cases; in three of these the right base, and in one the right apex, was affected.

Delirium tremens complicated pneumonia in seven cases, and five of these were fatal.

Otorrhœa from tympanitis occurred three times, and pulmonary *embolism* once, with recovery.

Many other complications recorded, as tonsillitis, gout, asthma, cardiac and renal disease, and laryngitis, were no doubt accidental coincidences. None of these occurred more than four times among the 434 cases.

In probably every case there was pleurisy; but serous effusion was only abundant enough to be noticed in 17 cases.

The most frequent sequel was *empyema*, which followed pneumonia in 24 cases, one of them being circumscribed empyema of the right apex.

7. **Duration.**—This was measured by the pyrexia, which occasionally preceded the evidence of hepatisation, and more often ceased before the signs of consolidation had disappeared. In 118 cases the duration was not ascertainable.

The shortest cases lasted three days; and these mild, but certainly not "abortive," cases occurred in children or youths, as the following detailed statement shows:—

Duration of Pneumonia

Days.	Ages of Patients.	Cases.
Three	3, 5, 5, 5, 6, 8, 9, 12, 14, 22, 27	11
Four	5, 6, 7, 7, 7, 10, 11, 11, 12, 12, 13, 14, 14, 17, 19, 19, 20, 21, 21, 22, 26	21
Five		30
Six		68
Seven		62
Eight		39
Nine		28
Ten		20
Eleven		12
Twelve (in four recovery delayed by complications)		8
Thirteen (fall of temperature by lysis)		3
Fourteen		3
Fifteen		3
Seventeen		3
Twenty-one (delayed by complications)		3
Above three weeks (delayed by complications)		2
		316

The most frequent duration of the disease was about a week; 32 patients were actually ill for only three or four days, 199 (nearly two-thirds of the number) for five to nine days, and only 17 for more than twelve days.

8. **Mortality.**—The total number of deaths was 111, a high per-

centage (25·5) out of 434.[1] But this general statement is of little value. Hospital mortality in this as in most other diseases is higher than that of private practice, because the previous habits and conditions of the patient are less favourable. But again, general cases seen in consultation are usually severe, and sometimes hopeless. Of 73 of the latter class 25 were fatal—more than a third.

Deaths among 362 Hospital Patients (86)

Sex.	Age.	Highest observed Temp.	Herpes.	Locality.	Complications.
F.	47	103·4°	Absent	Right base	Ulcerative endocarditis and meningitis
M.	31	102·4	,,	Double	...
M.	43	104	,,	Double	In course of phthisis
M.	49	103·2	,,	Double	In course of phthisis
M.	37	100·4	,,	Right apex	Carcinoma of stomach
M.	43	101·4	,,	Left base	Hæmoptysis ; gumma of liver
M.	27	100·2	,,	Left apex	In course of morbus Brightii
F.	12	102·8	,,	Double	Gangrene of lung ; chronic disease of spine
M.	22	102·2	,,	Right lung	Ulcerative endocarditis
F.	51	104·4	,,	Left apex	Bronchitis
M.	43	104·2	,,	Right apex	Gangrene of lung ; pericarditis
M.	49	...	,,	Double	In course of morbus Brightii
M.	10	100·2	,,	Right base	Chronic basal meningitis
M.	39	103	,,	Right base	Marked delirium
M.	2	104	,,	Right base	Laryngitis
M.	33	104·6	,,	Left apex	Pleuritic effusion
F.	29	100	,,	Right base	Cancer of bowel
M.	42	...	,,	Right apex	Morbus Brightii (lardaceous)
M.	45	...	,,	Left base	Morbus Brightii ; pericarditis
M.	66	...	,,	Right apex	Pericarditis ; delirium
M.	36	109	,,	Right base	Intemperate ; hyperpyrexia
M.	41	105·8	,,	Right apex	Intemperate
M.	35	103	Present	Left base	Delirium tremens
F.	46	103·6	Absent	Double	Gangrene of lung ; ulcerative endocarditis
F.	42	101·8	,,	Left base	Tumour of brain
M.	54	106·4	,,	Right apex	Intemperate; ulcerative endocarditis
M.	34	104·6	,,	Right base	Delirium
M.	39	...	,,	Left base	Delirium
M.	55	...	,,	Left base	Pleuritic effusion
M.	58	102	,,	Right apex	Pericarditis
M.	27	103·2	,,	Right base	Delirium
M.	25	102·4	,,	Right base	Pericarditis ; thrombosis of pulmonary artery
M.	33	103	,,	Left base	Delirium
M.	38	102·8	Present	Left base	Delirium tremens

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[1] The total mortality for all ages and both sexes, without for the most part exclusion of secondary cases, was 191 in 1060 (*Coll. Invest. Report*—private cases), 281 in 2618 (Huss—hospital and private at Stockholm), and 192 in 1010 (Coupland—Middlesex Hospital).

Deaths among 362 Hospital Patients—*continued*

Sex.	Age.	Highest observed Temp.	Herpes.	Locality.	Complications.
M.	42	104°·2	Absent	Left base	Intemperate
M.	36	105·8	,,	Double	Intemperate; gangrene of lung
F.	63	102·4	,,	Right base	Pericarditis
M.	49	104	,,	Left base	Intemperate; gangrene of lung
M.	27	107·8	,,	Left base	Hyperpyrexia
M.	21	102·4	,,	Double	Delirium
M.	33	104·4	,,	Right apex	Intemperate; pericarditis
M.	22	103·4	,,	Double	...
M.	39	97·8	,,	Double	Admitted 7th day, moribund
M.	8	104	,,	Double	Pericarditis; empyema
M.	22	103·6	,,	Left apex	Delirium tremens
M.	17	103·4	,,	Double	Admitted comatose
M.	47	103·4	,,	Left base	Diabetes
M.	48	102·4	,,	Double	Intemperate
F.	18	105	,,	Double	...
M.	4	104·6	,,	Double	...
F.	3	103·2	,,	Double	...
M.	27	102·6	Present	Left base	Endocarditis
M.	45	103·6	Absent	Left base	Influenza; pericarditis
M.	26	..	,,	Right apex	Delirium
M.	41	105	,,	Right apex	...
F.	21	105·2	,,	Double	Pregnancy; empyema
M.	29	103·4	Present	Right lung	Pericarditis
M.	15	100	Absent	Right lung	Hodgkin's disease
F.	58	104·2	,,	Left apex	Delirium
M.	48	103·6	,,	Double	...
M.	63	101·2	,,	Right base	...
M.	45	105·4	,,	Right apex	Pericarditis; delirium
M.	37	103·4	,,	Double	Delirium tremens
M.	52	103·6	,,	Double	Intemperate
M.	32	104·8	,,	Left base	...
M.	58	102·2	,,	Right apex	Acute nephritis
M.	44	102	,,	Left base	Chronic nephritis
M.	20	104·2	Present	Left base	...
M.	20	104	Absent	Double	...
M.	4 m.	104·4	,,	Right lung	Pericarditis
M.	33	104·6	Present	Double	Delirium; laryngitis
M.	22	103	Absent	Double	Laryngitis
F.	29	104·2	,,	Right lung	Intemperate
M.	62	101·4	,,	Left apex	Fatty liver
M.	36	102	,,	Left base	Intemperate
F.	18	105	,,	Right lung	Empyema
M.	48	101·4	,,	Right base	Pericarditis
M.	63	...	,,	Right base	Morbus Brightii
M.	57	...	,,	Right lung	...
F.	48	...	,,	Double	...
M.	35	...	,,	Double	Intemperate
M.	54	...	,,	Double	Endocarditis; meningitis
F.	2½	...	,,	Right apex	...
M.	56	104	,,	Left base	Laryngitis
M.	35	104	Present	L. lung and R. apex	Intemperate

Deaths among 73 Private Cases (25)

(*Herpes absent in all*)

Sex.	Age.	Highest observed Temp.	Locality.	Complications.
F.	39	103°	Left base . .	Intemperate ; pregnant
M.	27	101	Right base . .	Intemperate
M.	48	...	Right base . .	Intemperate
F.	30	105	Right base . .	Delirium
F.	15	104	Left base . .	...
F.	43	104	Double . . .	...
F.	66	103	Right base . .	...
F.	43	102·5	Left base . .	...
M.	58	98	Left base . .	Chronic Bright's disease
F.	40	105	Left base . .	Pericarditis
M.	72	101	Right base . .	Phthisis
F.	45	104	Right lung . .	Icterus
M.	42	104·4	Right base . .	...
F.	45	108·5	Right base . .	Puerperium
M.	57	103	Left base . .	...
M.	58	102	Left base . .	Diabetes
F.	68	104	Right apex . .	General bronchitis
M.	45	103·5	Right base . .	Intemperance
M.	75	101·5	Right base . .	Aortic valvular disease
F.	55	104	Right base . .	General bronchitis
F.	75	104·5	Right mid-lung .	Delirium
M.	62	102·5	Right lung . .	Emphysema
M.	66	104	Left base . .	...
M.	20	104	Double . . .	Mania
M.	49	106	Left base . .	Delirium

Death-rate of Pneumonia at several Ages

Under 5,	5 deaths	out of	20 cases.	Four months (pericarditis), 2½, 3 (double), 2 (laryngitis), 4 (double).
Between 5 and 10,	1 death	,,	66 ,,	8 double, with pericarditis.
,, 10 ,, 15,	2 deaths	,,	45 ,,	10 (meningitis), 12 (spinal disease).
,, 15 ,, 20,	5 ,,	,,	48 ,,	15 (general bronchitis), 15 (Hodgkin's disease), 17 (double), 18 (double), 18 (empyema).
,, 20 ,, 25,	9 ,,	,,	45 ,,	20 (double), 20, 20 (double), 21 (double), 21 (double), 22 (intemperance), 22 (double), 22 (endocarditis), 22 (double).
,, 25 ,, 30,	10 ,,	,,	38 ,,	25 (pericarditis), 26, 27, 27 (endocarditis), 27 (intemperance), 27 (tubal nephritis), 27 (hyperpyrexia), 29 (intemperance), 29 (cancer), 29 (pericarditis).
,, 30 ,, 35,	8 ,,	,,	29 ,,	30, 31 (double), 32, 33, 33, 33 (double), 33 (intemperance), 34.

Between 35 and 40, 13 deaths out of 33 cases.	35 (double), 35 (intemperance), 35 (double, intemperance), 36 (intemperance), 36 (intemperance), 36 (intemperance), 37 (cancer), 37 (double, intemperance), 38 (intemperance), 39 (double), 39 (intemperance), 39, 39.
,, 40 ,, 45, 13 ,, ,, 27 ,,	41 (pericarditis), 41 (intemperance), 42, 42 (intemperance), 42 (morbus Brightii), 42 (tumour cerebri), 43, 43 (syphilis), 43 (double) *bis*, 43, 43 (pericarditis), 44 (morbus Brightii).
,, 45 ,, 50, 18 ,, ,, 27 ,,	45 (pericarditis), 45 (pericarditis), 45, 45 (intemperance), 45 (hyperpyrexia), 45 (morbus Brightii), 46 (endocarditis), 47 (endocarditis and meningitis), 47 (diabetes), 48 (pericarditis), 48 (double) *ter*, 48 (intemperance), 49 (morbus Brightii), 49 (intemperance), 49 (double), 49 (hyperpyrexia).
,, 50 ,, 55, 4 ,, ,, 5 ,,	51 (bronchitis), 52 (double, intemperance), 54 (endocarditis) *bis*.
,, 55 ,, 60, 10 ,, ,, 15 ,,	55 (bronchitis), 55, 56 (diphtheria), 57, 57, 58 (morbus Brightii), 58 (pericarditis), 58 (double), 58 (diabetes), 59.
,, 60 ,, 65, 5 ,, ,, 12 ,,	62, 62, 63 (pericarditis), 63, 63 (morbus Brightii).
,, 65 ,, 70, 4 ,, ,, 6 ,,	66, 66, 66 (pericarditis), 68.
Over 70, 3 ,, ,, 5 ,,	72, 75, 75 (morbus cordis).

Causes of death.—That age is a grave element in the prognosis of pneumonia is clear from the last table. Excepting infants, there are few deaths under 15. After this age, the mortality rises steadily with the age of the patient, although even after 70 the prognosis is not always fatal. It will also be noted that the deaths at the earlier ages are generally accompanied by one of the untoward complications to be mentioned presently; whereas most of the fatal cases in the later decades are uncomplicated.

In all cases, single or double, and at all ages, the important causes of death were as follows:—

Intemperance, with or without delirium tremens, was present in a marked degree in 16 of the fatal cases, and this is probably below the truth.

In 10 other fatal cases delirium, more violent and particularly more diurnal than usual, was noted; and although only 4 cases were distinguished as well-marked delirium tremens, many of the other delirious patients were intemperate.

Pericarditis occurred in 14 cases, all of which proved fatal: endocarditis in 7, and meningitis in 5.

Bronchitis was fatal in only 3 cases, all old people.

In 3 fatal cases there was pleuritic effusion, and in only 1 of these was it purulent; so that all the cases followed by empyema without other complications ended in recovery except this one.

Hyperpyrexia—a temperature over 106°—occurred in 6 patients, of whom 2 recovered (under 107°), and 4 died.

Of fatal cases of *secondary* pneumonia 8 occurred in the course of Bright's disease; 5 in course of chronic tuberculosis; 2 in diabetes, and the 6 others happened in cases of cancer, lymphadenoma, tertiary syphilis, cerebral tumour, and valvular disease of the heart.

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Catarrhal Pneumonia

Synonyms.—*Pulmonary catarrh, Broncho-pneumonia, Lobular pneumonia, Disseminated pneumonia,* including *Vesicular pneumonia.*

Definition.—This disease is more difficult to define than is lobar pneumonia. In some cases it closely resembles the latter both clinically and anatomically, while in others the two diseases offer few points of resemblance. Lobular pneumonia, as an anatomical lesion, includes such different conditions as acute pulmonary catarrh in a child after measles, the chronic broncho-pneumonia of rickets, the hypostatic pneumonia of fever (in part), the caseating pneumonia of phthisis, and the lobular suppuration of pyæmia. The last two conditions form part of the pathology of tuberculosis and of septic embolism respectively. Hypostatic pneumonia is a secondary condition probably beginning in the bronchi, certainly secondary to prolonged passive congestion, and made up of lobular hepatisation, often confluent and sometimes primarily lobar, with œdema, and collapse. The remaining kinds of pulmonary inflammation which are included under the head of Catarrhal Pneumonia are better dissociated from lobar or fibrinous pneumonia, and named acute or chronic pulmonary catarrh.

Of the disease thus restricted we may say that it befalls children far more frequently than adults; that, though sometimes acute in its origin, the signs of its invasion are never so abrupt or well marked as those of lobar pneumonia; that its course is either subacute or chronic, and that it never terminates by crisis; that it is accompanied by bronchitis, and is probably always secondary. It is much more directly connected with breathing cold air, or air laden with dust and other mechanical impurities, than is lobar pneumonia, and seldom occurs in an epidemic form. It is often secondary, not only to bronchitis, but also to measles and whooping-cough, and to chronic conditions of ill health, particularly rickets. As a rule it affects both lungs, and that in irregular patches, without preference for either apex or base. Although often accompanied by pleurisy, this may be absent.

Anatomy.—As one of its names implies, catarrhal pneumonia affects the lungs lobule by lobule, and these lobules are often found scattered over both lungs, whence the name "disseminated pneumonia." More often several adjacent lobules are affected together so as to form patches in different parts of the lung. These patches again may coalesce, and thus after death from pulmonary catarrh a considerable area of continuous solid and hepatised lung is found which closely resembles the lobar hepatisation of fibrinous pneumonia. Pulmonary catarrh does not affect the base more frequently than the rest of the lung; nor is there reason to believe that it has a predilection for the apices, except when it is part of the effects of tuberculous invasion. Occasionally only part of a lobule may be affected; smaller, separately inflamed areas may, in some cases, be distinguished by the naked eye, and this kind of disseminated pulmonary catarrh may be distinguished as "vesicular pneumonia."

Microscopic examination shows that the disease originates in a catarrhal inflammation of the mucous membrane of the bronchi; this spreads to the last bronchioles, which open into the lobules—a condition described as "capillary bronchitis." When the lobule is itself examined it is found filled with epithelium and with smaller cells which have the characters of leucocytes. There are no blood-discs, and fibrin is absent or scanty. The larger epithelioid and smaller inflammatory cells fill the air-vesicles and intralobular passages, and as they accumulate expel the air and convert each infundibulum, and at last the whole lobule, into a small solid mass.

Along with hepatised lobules there are almost always found, particularly in young children, some lobules which have undergone collapse; they are airless, but not soft; shrunken, not swollen; and empty, that is, not stuffed with inflammatory products. These collapsed portions are seen as depressed, dark patches on the surface of the lung, and particularly at its lower edges. They were formerly confounded with the patches of lobular pneumonia which they so often accompany; but from these they are entirely distinct, and it is doubtful whether such collapsed portions of lung are capable of undergoing the process of lobular inflammation.

Bacteriology.—The encapsuled diplococcus (or pnemonococcus) of lobar pneumonia is often found in the alveolar contents of the affected lobules; but it is often absent, and with it or in its stead may be seen various other micrococci. There is no reason to believe that pulmonary catarrh depends upon, or even is constantly associated with the presence of any one specific microbe.

Ætiology.—Catarrhal lobular pneumonia is almost always associated with bronchitis, and is usually secondary to it. It is in children often accompanied with signs of rickets; and it is a frequent sequel of measles and whooping-cough, less frequently of scarlatina, small-pox, or enteric fever. It may follow as the result of burns, and complicate infantile "dysentery." Why it is comparatively rare in adults is difficult to say. It does not appear to be a specific infective disease, and its relation to true lobar or fibrinous pneumonia rather obscures the ætiology of the latter than lights up its own.

Clinical symptoms.—The symptoms and signs of pulmonary catarrh are far less striking than those of acute lobar pneumonia. There are no rigors, no sudden rise of temperature, to mark its onset; we suspect rather than ascertain its presence when a case of bronchitis in a child or an aged patient is accompanied with fever; and comparatively slight physical signs are sufficient to confirm our suspicions.

The onset of pulmonary catarrh is gradual; the fact that bronchitis is extending to the smaller tubes is not shown by any trustworthy physical sign, although we may find a sonorous, deep-toned rhonchus replaced or accompanied in certain parts of the chest by a high-pitched sibilus; or the large, coarse, toneless rattles produced by mucus and air in the trachea and larger bronchi may be replaced by mucous râles of the same quality, but smaller, that is to say with more numerous explosions in each inspiration. These physical signs may or may not be present. Our recognition of the presence of what is called capillary bronchitis depends upon the symptoms of dyspnœa—upon cyanosis, increased rapidity of breathing, and sucking in of the soft parts about the thorax with each inspiration. When inflammation has gone still farther and affected the lobules, there is often, as we should expect from the above anatomical account, no diminution of resonance on percussion; for the solidified lobules are scattered, and resonance spreads, while dulness does not. If several inflamed or collapsed lobules are near enough to form an airless patch, we may then recognise on light percussion a note shorter, of higher pitch and of diminished tone, compared with that afforded by the rest of the chest; but probably the earliest physical sign of the presence of lobular catarrh will be a consonating, more or less musical quality in the previously toneless inspiratory râle.

If the affected lobules become so numerous and close as to form a large solid area in one part of the lung, we obtain more decided dulness on percussion; and the râles become finer and more consonating until they approach very nearly the characteristic fine crepitation of lobar pneumonia. These last signs are not only slow in appearing, or absent throughout the whole case, but they do not advance steadily from the base upwards. They appear most often in the middle of the back, internal to one or the other shoulder-blade, sometimes at one base, sometimes in the armpit, and occasionally at the apex; but they frequently shift in position, and are very seldom symmetrical on the two sides. This absence of definite local distribution is an important element in distinguishing catarrhal from fibrinous pneumonia, and also from phthisis; but unfortunately it affords no evidence against the presence of diffused pulmonary tuberculosis.

In a further stage of consolidation the dulness may be as marked and extensive as in lobar pneumonia. Tubular breathing will then be heard, and marked bronchophony, not with the child's ordinary voice but with its cry.

The pyrexia which shows the presence of lobular and vesicular pneumonia is usually moderate in degree, even in children; and rarely approaches the height seen in lobar pneumonia, in septicæmia, or in tuberculosis. The course of the fever is irregular without constant evening accessions, but remittent rather than intermittent.

The skin of a patient suffering from catarrhal pneumonia is often dry and hot, but it never has the pungent feel characteristic of fibrinous pneumonia; and in many cases, especially in children, it is covered with sweat. This is particularly the case when the broncho-pneumonia is tuberculous; but the symptom is not only inconstant in itself, it is greatly modified by the use of poultices, steam-kettles, and other modes of treatment.

The urine is often scarcely affected, and when febrile in other characters very seldom contains albumin.

The pulse is frequent and usually soft, more frequent in proportion to the temperature than in lobar pneumonia.

The respirations increase in frequency in proportion to the extent of the lungs involved. In mild cases they are not above thirty, but in severe cases rise to fifty, and even considerably higher. The forced muscles of respiration are usually brought into play, and the degree in which they are used furnishes another valuable evidence of the severity of the disease. The nostrils dilate with each inspiration, as in many of the lower animals; and the inspiratory movements of the diaphragm are followed by expiratory contractions of the muscles of the abdominal wall. In young children, of whom we chiefly speak in this description, respiration is mainly abdominal, and in dyspnœa the muscles which move the thorax have less play than in adults; but in these patients the want of resistance of the tissues causes much earlier and usually more marked movement of the soft parts than in adults. With inspiration a deep depression is seen to form above each clavicle, and another between the trachea and the manubrium. The epigastrium is drawn in, and even the lower ribs and ensiform cartilage yield to atmospheric pressure, particularly when the bases of the lungs are collapsed and airless.

Orthopnœa may be seen when extensive catarrhal pneumonia affects an adult, but is less common than in dyspnœa from cardiac disease, bronchitis, or lobar pneumonia; and in children it is decidedly less common than in dyspnœa from laryngitis or from empyema.

The important symptom of insufficient aeration of the blood, which consists of blueness of the surface, is present in all severe cases of pulmonary catarrh. It is usually first seen in the lips and the ears, then in the fingers and toes, next in the face, and finally over the whole surface of the body and the mucous membrane of the mouth. While the frequent and forced respirations show the want of air, the cyanosis just described shows how ineffectual these forced and frequent efforts are in supplying the want. Thus the rapidity of the pulse indicates the increased efforts of the heart to keep up the pulmonary circulation, and the paleness of the skin proves the deficiency of supply to the systemic capillaries, and gives us a hint of the similar failure of circulation in the lungs.

In adults affected with catarrhal pneumonia the expectoration is usually scanty, and consists of thin mucus without much admixture of air. In most cases, however, there is already present the frothy mucopurulent expectoration of precedent bronchitis. The rusty sputum of fibrinous pneumonia, whether prune-juice, or saffron, or greenish in tint, is probably never seen in cases of lobular pneumonia. In cases with the symptoms and course of catarrhal, not fibrinous, pneumonia, I have occasionally observed scanty and nearly airless sputa, of a pinkish colour from the presence of blood; and streaks of blood probably derived from the veins of the trachea or upper air-passages may sometimes be seen, as in ordinary cases of bronchitis which do not extend to the lobules. True rusty sputa are as nearly pathognomonic of fibrinous pneumonia as any symptom can be; and pure hæmoptysis under similar circumstances is almost as characteristic of phthisis; but the expectoration offers us no help in the often difficult question between uncomplicated pulmonary catarrh and disseminated tuberculosis of the lungs.

In children, not only in infants, but often up to the age of eleven or twelve, there is a remarkable inability to expectorate. Phlegm is coughed up into the larynx and then swallowed; and though some children as young as seven or eight (once a boy of only five) have learnt how to get rid of it, and others may be taught the art, we must reckon upon the absence of this valuable help in diagnosis. When by natural or forced vomiting a child empties its air-passages of accumulated secretion, mucus and muco-purulent matter may be seen in the vomit; some of it perhaps lately swallowed, and some ejected directly from the larger bronchi. In such cases the presence or absence of rusty sputa may be observed.

In aged patients it is not uncommon to find the same inability to expectorate as in children.

As the disease goes on, the patient's appetite suffers; he becomes pale and thin; his nights are constantly disturbed by cough, and his strength gradually fails.

Course, complications, and prognosis.—The duration of acute pulmonary catarrh is undetermined. Infants may die in a few days from want of power to get rid of the secretion which obstructs the air-passages; and in aged persons catarrhal pneumonia, or so-called capillary bronchitis, often occurs as the last stage of chronic bronchial catarrh, and proves fatal in two or three days. But, with the exception of the two extremes of life, patients suffering from catarrhal pneumonia seldom die within a week or even a fortnight from the date of recognition of the disease. Most often its special symptoms and signs gradually disappear, and the condition of simple bronchitis in which the disease began remains at its conclusion. In children, particularly, this also gradually subsides, as a rule, and complete convalescence is established.

Of the complications to be feared, the most frequent and formidable is tuberculosis, either in its generalised form or in that of chronic phthisis. A large proportion of cases of bronchitis and broncho-pneumonia in children are associated with the presence of caseous lymph-glands, cervical, mesenteric, or mediastinal; and the bacilli which already exist in the lympharia may readily infect the lungs and other organs.

Another important complication of catarrhal pneumonia in children is empyema, and this must be sedulously looked for, or it may escape notice. In infants the most frequent complication is extensive collapse of the lung, which, as above stated, almost always accompanies lobular pneumonia at an early age, and often determines a fatal event.

On the whole the prognosis is most affected by the age of the patient. Broncho-pneumonia in little children and broncho-pneumonia in the aged are very fatal diseases. In children above two or three years old the forecast is much better, but it is still decidedly worse than in cases of lobar pneumonia occurring at the same age. In adults uncomplicated pulmonary catarrh is rare, and usually dependent upon some special form of irritation. In such cases the prognosis depends upon the nature of the irritant and the probability of its being withdrawn.

Pulmonary catarrh, which is secondary to measles or whooping-cough, is more serious than that which occurs without these precedents. Again, when it occurs, as is so often the case, in a child affected with rickets, it is more likely to be intractable or fatal than when this condition is absent. Of the symptoms of the disease, the extent of lung involved in inflammation or collapse, the frequency of the pulse, and above all the degree of dyspnœa estimated by the symptoms above detailed, are the most important elements of prognosis.

The most dangerous symptoms are those of suffocation, and this is the most frequent immediate cause of death.

Next in importance to the lividity and the forced and rapid breathing, which show pulmonary obstruction, are the weak and frequent pulse and pallor which point to failure of the heart.

In little children pulmonary collapse is often as extensive as pulmonary catarrh. After the second year is passed, this complication is less frequent and dangerous, and in adults it scarcely occurs except at the posterior edge of the lower lobe. The most serious complication of all is the presence of tubercle.

Diagnosis.—The recognition of broncho-pneumonia in most cases is not difficult; but certain mistakes are apt to occur, and in some cases it is impossible to decide upon the exact nature of the pulmonary inflammation present, except by the progress of the case.

From lobar pneumonia the diagnosis, founded on the onset and course, as well as on the symptoms above enumerated, is only difficult when many inflamed lobules coalesce to form a single large patch towards the base of the lung. Here dulness on percussion, tubular breathing, and crepitant râles will simulate primary lobar inflammation; and when such a case is seen for the first time it is almost impossible to avoid the mistake. In children we have not the help afforded by the sputum, and the physical signs are not so definitely localised as in adults.

On the other hand, true fibrinous pneumonia in children is sometimes called lobular merely because of the patient's age. The shortness of its course will correct the error. In elderly people the difficulty is to distinguish broncho-pneumonia from "capillary" bronchitis—from bronchitis, that is, affecting the smallest bronchioles, but leaving the lobules free. Probably the two affections are often present together. When rhonchus and sibilus are present over the whole of both lungs, with little or no elevation of temperature, and with marked cyanosis, it is unlikely that the bronchitis is complicated by pneumonia. When one lung is decidedly more affected than the other, and particularly if partial dulness, bronchial breathing, or crepitation appear in patches which shift their position, and if these symptoms are accompanied by pyrexia, we may be sure that lobular pneumonia is present.

The most common and important difficulty of diagnosis is between lobular pneumonia and acute tuberculosis of the lungs—not phthisis, for in its symptoms and localised physical signs this disease is almost always characteristic; if it be overlooked or mistaken in its early stages, it is not confounded with lobular pneumonia. No doubt, a chronic broncho-pneumonia confined to one apex would be difficult or impossible to distinguish from early phthisis; but the existence of such an affection, apart from the actual presence of tubercle, is extremely rare in an adult. Acute broncho-pneumonia of one apex with pyrexia, frequent pulse, sweating and general bronchitis, may, however, closely simulate phthisis in a child. When the previous condition and mode of onset are unknown, it is difficult if not impossible to distinguish this from phthisis; in fact, the physical signs are the same. The far more frequent difficulty is to decide upon the presence of disseminated tubercles in the case of children who do not throw off a pulmonary catarrh, but week after week grow paler and thinner and more feverish; or, again, in the case of adults who, long subject to bronchitis, begin to show the pyrexia and physical signs of broncho-pneumonia in addition. In such cases the bronchitis and lobular pneumonia are actually present whether tubercle be there or not. The presence of the latter must be recognised by other than auscultatory signs. In adults there is no likelihood at this stage for blood and nummular sputa and fragments of elastic tissue to be present in the sputa, but we may find the bacilli of tubercle—a discovery which at once decides the question. In children this means of diagnosis is absent; and we depend rather on the height and irregularity of the pyrexia, on the rapidity and degree of emaciation, and the amount of sweating. The same symptoms help us in the recognition of acute miliary tuberculosis in an adult. In the latter case, however, the question more often lies between tuberculosis and bronchitis.

A disease which not infrequent mistakes warn us to be watchful against confounding with broncho-pneumonia in children, is empyema. In both cases we have pyrexia, dulness on percussion, and cough, with dyspnœa and without expectoration. In both the child may be pale and thin, and the fingers clubbed; in both its voice may be too weak and high pitched to help us by yielding bronchophony or tactile fremitus; and, lastly, the small area of an infant's chest, the loudness of its breathing, and certain conditions which favour conduction of bronchial breathing through effused liquid, even in an adult, may all combine to obscure the diagnosis of empyema. On the other hand, a knowledge of this danger will sometimes lead even an experienced physician to suppose that dulness at the base of the lung with no vocal resonance or crepitation must be due to pleuritic effusion, whereas it is really a patch of inflamed and collapsed lobules.

The height of the temperature is often a guide, but, on the one hand, this does not distinguish empyema from broncho-pneumonia associated with tubercle; and on the other hand we sometimes, though rarely, meet with empyema in which, even in a child, the temperature is scarcely above normal. One help we may gain in doubtful cases by listening to the chest when the child is crying; we may then often obtain both bronchophony and fremitus; and, after a prolonged scream, so deep an inspiration is taken that crepitation or tubular breathing, before unheard, becomes distinctly audible.

In doubtful cases of the kind the use of a grooved needle or a hypodermic syringe is most valuable. It will decide a question which cannot be settled by the most careful and repeated auscultation; and this is its only legitimate use.

Beside tuberculosis, another general disease is not infrequently mistaken for broncho-pneumonia, both in children and adults, namely, enteric fever. Here we have pyrexia, often of irregular course, and usually, sooner or later, accompanied with pulmonary congestion, bronchitis, or hypostatic pneumonia. The bowels are not infrequently constipated, an enlarged spleen cannot always be felt, and in children the characteristic rose spots are sometimes absent. When seen for the first time, and with an imperfect history of the case, a decision is sometimes impossible. The points to look for are the early or later occurrence of pyrexia or cough, the course of the temperature, the presence of headache, delirium, or apathy, and the fulness of the abdomen. If enterica be present, a few spots, after repeated searches, will generally be found on the back and loins, if not on the abdomen and flanks; and repeated trial will seldom fail to decide whether the spleen be enlarged or not. In doubtful cases pulmonary catarrh is more probable in the case of children, and enteric fever in the case of adults.

Treatment.—In the early stage of pulmonary catarrh the treatment is that of bronchitis. We endeavour to relieve cough, pain, and oppression of the chest, to promote secretion in the affected parts, and to favour action of the bowels, the kidneys, and the skin. For this purpose confinement to bed is usually desirable; but with little children it is sometimes better to cover the chest with a cotton-wool jacket, and allow them to lie in the nurse's arms, or to sit up when they cough. In cold dry weather a steam kettle is a useful help in addition to an open fire; and an adult patient will find still greater relief by inhaling steam from boiling water to which compound tincture of benzoin, eucalyptus, turpentine, terebene or some other aromatic oleo-resin has been added. When the air is warm and moist there is no object in making it moister. The same applies to protection by curtains, screens, canopies, and tents. In the winter season, in large wards or draughty rooms, these appliances are most valuable; but when there is too free a secretion of mucus, when the patient is sweating, feverish and restless, a close, hot and damp air is not the best for the patient to breathe.

As soon as febrile symptoms have subsided and the patient may be considered convalescent, it is most important for him to breathe the open air when the weather is at all suitable. Even in winter a child well wrapped up and carried out for a quarter of an hour at a favourable time will often show by improved appetite and better sleep the benefit of fresh air. This is particularly important in the case of children suffering from broncho-pneumonia after measles or whooping-cough.

When the temperature is high and the skin is very hot, tepid sponging is called for, and the child should be put in a warm bath every evening. If fever should run high, repeated lukewarm baths are the best means of reducing it. A mustard plaster on the front of the chest is often extremely useful in the early stages of the disease; afterwards a jacket poultice of linseed is a common and for the most part a good application: it relieves pain, promotes action of the skin, moderates the cough, and comforts the patient. For infants, however, its weight and tightness are as a rule undesirable, and it may be better replaced by a jacket of cotton-wool worn next the skin.

The food of a patient with broncho-pneumonia should be liquid, given in comparatively small amount and more frequently than in health. To young and weakly children a little milk or broth should be given every two hours; but in other cases there is no need for such frequent feeding, and four hours is not too long a time to elapse between each meal; even this is sometimes too short an interval for the digestive powers of an older patient, and it is better to be content with three or four meals in the day.

Diluents should be taken freely; cold water, soda water with milk or fruit syrup, thin barley-water, toast and water, tamarind water, or lemonade made with cream of tartar; this "imperial drink" is grateful to the patient, and is also diuretic and slightly laxative.

Stimulants are not to be prescribed as a matter of routine. Brandy should be given if the pulse be very rapid and the action of the heart weak and irregular.

The most useful drugs in the early stages are ipecacuan, squill and nitre, sweetened with syrup of tolu or oxymel. Occasionally in the early stage of the attack a few drops of antimonial wine are efficacious in promoting secretion in the bronchial tubes and skin. In serious cases with urgent dyspnœa no drug is so valuable as carbonate of ammonia given in doses of one grain for an infant to five for an adult; its pungency may be covered by liquorice, treacle, or syrup.

When there is marked cyanosis, with a small and weak radial pulse and distension of the jugular veins and epigastric pulsation, bleeding to six, eight, or even ten ounces is indicated; and may often save a patient's life. In the case of children, two or three leeches on the sternum may be applied in similar circumstances.

As soon as the temperature is normal and the physical signs abated, the patient should be removed to another room, and allowed to sit by an open window, if the weather be favourable. Beside drives in an open carriage, it is often desirable that removal to the south coast of England or to the shores of the Mediterranean should follow an attack of bronchitis with broncho-pneumonia. Children should be removed as soon as possible to the seaside, or at least to pure country air.

CHRONIC PNEUMONIA

It is doubtful whether acute lobar pneumonia ever ends in a chronic inflammatory process. At any rate, in the great majority of cases, if the patient recover, the inflamed lung recovers also, and completely. I once had the opportunity of observing the state of a lung in a patient who died accidentally about a fortnight after recovery from acute fibrinous pneumonia. The previously inflamed part of the organ was still distinguishable; its consistence was firmer, its colour darker, and it contained less air and more serum than the rest of the lungs: but no other traces of hepatisation were present, and it was no doubt functionally active.

Chronic lobar hepatisation.—Addison, however, described a condition of persistent consolidation of lung following acute lobar pneumonia, in which the section is no longer soft and granular but homogeneous, smooth and tough; though still solid and airless. He believed that recovery from this condition might take place. He called it "uniform albuminous induration" (1*a*, p. 28). Charcot and some other modern writers admit the existence of a similar condition, but regard it as a coalescence of inflamed lobules; and due therefore to catarrhal, not fibrinous pneumonia (7). We cannot doubt the existence of an anatomical state such as is described; but it must be extremely rare, and its true nature, origin, and event are at present undetermined.

Chronic broncho-pneumonia.—That lobular pneumonia may pass from a sub-acute to a chronic form is no doubt true if we regard the question of time alone without reference to acuteness of symptoms; but there is no evidence that an inflammatory process of a catarrhal kind will continue and spread after the temperature has become normal. The broncho-pneumonia which follows measles, whooping-cough, or diphtheria in children, and the much rarer broncho-pneumonia of adults, or that again which is caused by inhalation of dust (pneumoconiosis), have, so far as at present known, only one event if the patient neither dies in the acute stage nor recovers; and that event is infection by the bacillus of tubercle and the establishment of pulmonary phthisis.

Chronic interstitial pneumonia.—There is, however, another form of disease of the lung to which the name of chronic pneumonia is often given. It is that which was described by Cruveilhier as "*induration ardoise*," by Addison as "iron-gray consolidation" (1*a*, p. 28), by Corrigan as "cirrhosis of the lung" (8), by some French writers as "*sclérose pulmonaire*."[1] The origin and limits of the disease are still matters of dispute, and its clinical history does not appear to be precisely correlative with its anatomy. On the one hand, it has often been confounded with the more chronic forms of phthisis; on the other, it is usually associated with dilatation of the bronchi, although the two conditions are not always coincident. It appears sometimes to be a sequel of bronchitis or broncho-pneumonia, and sometimes to begin in successive attacks of pleurisy. Lastly, a similar anatomical condition is occasionally found in cases of syphilis, associated with gumma and probably originating in specific peribronchitis of the trachea and bronchi; this last group of cases is precisely analogous to those of so-called syphilitic cirrhosis of the liver. Clinically it may simulate (tuberculous) phthisis, and was formerly described as a variety of phthisis. The fact that this form of lues does not affect the apices of the lung and thence travel downwards, that it is not associated with other tuberculous lesions and is with those of syphilis, and the absence of bacilli from the sputum are the chief diagnostic points which usually guide us aright, even when hectic, emaciation, hæmoptysis, and the phthisical signs of phthisis are most misleading (*Path. Tr.* 1877, p. 313).

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[1] Bayle probably described this condition (as others have done since) under the title of phthisis with melanosis: Auenbrugger earlier still as scirrhus (that is, induration) of the lung. Corvisart and Chomel also described its anatomy independent of phthisis.

The resemblance, however, which Corrigan justly remarked between his chronic indurating fibrous process in the lung and that which was described by Laennec as cirrhosis of the liver, is an anatomical one. Anatomically we may put the two conditions together, and may compare with them the chronic interstitial nephritis of Bright's disease, which is often styled cirrhosis of the kidneys. We may even extend the comparison to the chronic indurating process in the nervous centres which is now named sclerosis; but in their origin these similar anatomical results differ greatly. By far the most frequent and characteristic forms of cirrhosis of the liver are due to intemperance, but there is no corresponding alcoholic cirrhosis of the lungs.

Morbid anatomy.—Pulmonary cirrhosis is most often limited to a single lung. It may begin in any part; but most frequently it starts from the root of the lung and spreads along the peribronchial connective tissue so as to cause on section a radiating appearance of fibrous bands. In other cases it spreads inwards from a patch of local pleuritic thickening. Occasionally it affects the base or the whole lower lobe, or the middle lobe of the right lung; while the rest of the organ remains unaffected. The new fibrous tissue is white, dense, and often so extremely tough as to cut like tendon, or even like cartilage. It is sometimes confined to broad septa or patches, leaving the rest of the lung free; but often it penetrates extensively between the lobules, mapping them out and giving a marbled aspect to a section. The pulmonary tissue itself is darker than the healthy parts of the lung, and varies from a slate colour to an almost black tint. It is firm, and contains less air than usual, but does not sink in water. On microscopical examination, the fibrous tissue is as dense as that of a tendon, though the course of the fibres is less parallel. The pulmonary tissue within a lobule is altered by collapse of some of the air-vesicles, by the lining epithelium being more visible and thicker than normal, by the capillaries being more or less obliterated, and by the thickening of the alveolar wall.

The process is not, therefore, a purely interstitial inflammation as defined by Virchow: it is parenchymatous also. Some pathologists, indeed, consider the intralobular changes as primary and essential; and the interlobular and peribronchial fibrous growth as secondary. But if it be true that pulmonary cirrhosis is seldom the consequence of bronchopneumonia, and most often takes its rise in peribronchitis or pleurisy, it seems probable that the primary seat of the disease, as of the corresponding changes in the liver, the kidneys, and the spinal cord, is in the interstitial connective tissue.

The affected lung on section shows cavities which can readily be traced to the bronchial tubes, of which they are certainly dilatations. These saccular pouches were regarded by Corrigan as the result of traction of the cicatrising fibrous tissue; and, although an opposite opinion has been ably defended by other pathologists, from Laennec downwards, it seems probable that the Dublin physician was correct. There are, no doubt, many cases of primary bronchiectasis, such as those which follow whooping-cough or bronchitis in children, and lead to uniform cylindrical dilatations without consequent fibrous thickening; and, again, such as form the bronchial pouches which have been described as retention-cysts in fetid bronchitis. The saccular dilatations in cirrhosis are often extremely irregular, and in some parts so closely packed together that scarcely any pulmonary tissue is left between them.

The result is contraction with diminution in bulk of the affected parts of the lung. There may be emphysema in the parts unaffected by cirrhosis, and sometimes large subpleural bullæ are seen. On the whole, however, the process is that of contraction. The affected lung becomes smaller as well as denser; and, when one side only is affected, the opposite lung may be hypertrophied and the mediastinum dragged over by the diseased lung.

Beside bronchiectasis, emphysema is also very frequently present, and shows its characteristic signs during life and anatomical appearances after death. Sometimes the hypertrophic pleurisy which has been the starting-point of pulmonary cirrhosis is only part of a general chronic inflammation with thickening of the whole pleuro-peritoneal cavity; and lungs, heart, and abdominal viscera are all affected by a similar process (*Path. Tr.* 1882, p. 172).

Symptoms.—These are often obscure and difficult of interpretation. They are chiefly of a physical kind. More or less dulness on percussion will be present, due probably to thickened pleura rather than to the pulmonary cirrhosis itself. Bronchial breathing may be heard, although this is far from constant; more often the pulmonary murmur is obscured by rhonchus and sibilus; but perhaps the most frequent auscultatory sign is the presence of râles, medium or large, and sometimes gurgling, accompanying inspiration and expiration. Not infrequently deficiency of breath-sounds, combined with the dulness, may raise a doubt whether there be an effusion of pus or serum in the pleura.

Expectoration is commonly abundant, muco-purulent in quality, and often nummular. Hæmoptysis is not unknown, even when cases of chronic phthisis are carefully excluded.

On inspection the affected side moves less freely than the sound one, and is ascertained by the cyrtometer to be the smaller. Owing to the same process of contraction the cardiac impulse may be displaced towards the affected side, or may be higher than usual.

There is often no pyrexia; the temperature is never high unless, as occasionally happens, septicæmia ensues from ulceration of dilated bronchial cavities. There is dyspnœa, increased on exertion, and the fingers may be clubbed.

Natural history.—Cirrhosis of the lung may come on at any age; but the majority of the patients are under fifty. It is rare in children, but not very rare between 15 and 20. The disease is more common in men than in women. It is often associated with intemperate habits, and sometimes with a similar interstitial fibrosis, degeneration, and shrinking of the kidneys or the liver; or with chronic peritonitis as well as pleurisy.

Diagnosis.—Some definitions of this disease would include all cases in which the physical signs denote a contracted, indurated, and comparatively airless condition of one or both lungs, due to fibrous degeneration of the pulmonary tissue, with the presence of numerous cavities containing pus and mucus. In the majority of such cases, however, this condition is due to tuberculous infection. The tuberculous disease affects both lungs; it begins in the apices and travels downwards. The cavities are excavations due to ulceration—vomicæ in the technical sense of the word. In fact the disease is chronic, and sometimes obsolete, tuberculous phthisis. Many of the earlier cases recorded by Andral, Corrigan, and Addison were undoubtedly tuberculous; and the same criticism applies to a majority, at least, of the cases which have been described by the late Sir Andrew Clark and other writers as "fibroid phthisis." The long controversy as to the degree in which the names pulmonary tubercle and phthisis are coextensive in signification may now be regarded as settled; and the final verdict is in favour of the doctrine originally taught by Laennec, and against that which distinguished between tuberculous and non-tuberculous phthisis. All phthisis is tuberculous; but, along with tubercle, catarrhal pneumonia, congestion, ulceration, bronchitis, pleurisy and fibrosis are always present. Phthisis of rapid course, with extensive ulceration and congestion, may still be called "pneumonic"; and cases which are long protracted, and perhaps at last cured, with abundant cicatrisation, may still be called "fibroid," or rather "fibrous"; but these are forms of essentially the same disease.

If all cases of cirrhosis of the lung were, as the late Dr. Moxon put it, "phthisis in the præter-pluperfect tense," there would be no need for the name; or, if retained at all, it would merely denote an anatomical condition present in various degrees in all cases of chronic phthisis. There is, however, abundant evidence that cirrhosis may be independent of tubercle from beginning to end; and the problem is to distinguish this non-tuberculous disease from the much commoner cases of chronic phthisis which simulate it, as well as from the fibrous degeneration which accompanies syphilis of the lung.

In the first place, phthisis almost always affects both lungs, cirrhosis is as a rule confined to one. Either disease may follow chronic bronchitis or repeated pleurisy; but in phthisis other organs, sooner or later, partake in the disease. Accordingly the presence of laryngitis with hoarseness or aphonia, diarrhœa, symptoms of tubercle of the testes or kidneys, of joints or of lymph-glands, is good evidence that the disease of the lung, however chronic, is tuberculous phthisis. Cirrhosis, on the other hand, is confined to the chest. It is a purely local condition; and its symptoms, in nature and degree, depend entirely upon the physiological effects of the local lesion. No doubt the diagnosis, comparatively easy as it is in the dead-house, is sometimes difficult at the bedside; but the presence or absence of the bacillus of Koch should be decisive.

Next to chronic phthisis, empyema is perhaps the affection most likely to be confounded with cirrhosis of the lung. In both cases the symptoms may be similar, namely, cough, wasting, pallor, pyrexia, dyspnœa; and in both there may be dulness at the base of one lung. In empyema the breath-sounds are usually absent over the dull area, and there may be no expectoration; but bronchial breathing is occasionally heard through pleuritic effusion, and if an empyema have opened into the lung, the expectoration may be of much the same kind as that which, in a case of cirrhosis, proceeds from a bronchial pouch. Moreover, in empyema some amount of contraction of the affected side of the chest is often present. The physical conditions are so similar that it is not surprising to find the physical signs also similar. The diagnosis depends, in most cases, upon a knowledge of the origin and progress of the patient's illness. In this, as in so many other cases, a right decision does not depend upon a single so-called pathognomonic symptom, but upon a wide survey of probable alternatives, and weighing of the course and probabilities of the individual case along with the actual physical signs present.

After all, in some cases puncture alone can decide the matter, and the test is readily applicable.

Prognosis.—Cirrhosis of the lung is always a grave but rarely a hopeless condition. The forecast varies with the amount of lung involved, with the duration of the disease, and, most of all, with the degree of general disturbance; loss of appetite, anæmia, wasting, sweating, vomiting, or diarrhœa are unfavourable circumstances. When the patient's weight is kept up, and he eats and sleeps well, we may hope that even extensive cirrhosis of the lung may gradually lead to contraction and obliteration of cavities, and final cicatrisation of the affected parts with hypertrophy of the opposite lung. Such a complete cure is no doubt exceptional; more often the disease passes into a permanently chronic condition, and the patient dies at last from bronchitis affecting the sound lung, or from some intercurrent affection.

Among the complications to which the patient with cirrhosis is liable may be mentioned—first, dilatation of the right side of the heart and anasarca; next, septicæmia from ulceration of one of the bronchial cavities; more rarely, abscess of the brain from pyæmia of a similar origin; or lardaceous disease, the result of prolonged suppuration.

Treatment.—From the nature of the case, the treatment must be tentative and expectant, following the indications of the patient rather than of the local disease. We endeavour to keep the expectoration from becoming fetid, and to check its amount, to relieve cough, particularly at night, by paregoric and other anodynes, and to hasten the process of cicatrisation by occasional counter-irritants, or by strapping the affected side with plaster. Inhalations of turpentine, thymol, terebene or creasote are often useful in lessening the secretion and correcting fœtor. At the same time, by help of mineral acids and bitters, particularly quinine and nux vomica, we try to improve the patient's appetite; with the same object we give him varied and abundant food, consulting rather his own caprice than ordinary rules of diet: of stimulants, by far the most useful, if the patient can bear it, is malt liquor, particularly porter. If this cause cough, constipation, or headache, ale or light German beer may be taken with advantage at the mid-day meal. Sometimes wine is better relished and proves more useful; in most cases it is certainly superior to alcohol in the form of spirits; if given at all, brandy is, as a rule, most useful when given as a sedative at night.

Whenever the weather permits it, the patient should be taken out of doors. When this is impracticable, he may sit before a widely open window, warmly wrapped up, and breathing through the nostrils with the mouth persistently closed. Fresh air often proves the most powerful promoter of appetite and of sleep.

Cases of cirrhosis of the lung are greatly benefited by climatic treatment; removal from dust-laden workshops and from foggy towns to pure air is the first step to improvement, and may often cut short the disease in its early stage. A mild and equable climate, such as that afforded by the south-west coast of England and many parts of Ireland, is the best for these patients.

There is no doubt that these cases are among those that derive most benefit by spending successive winters and springs on the Riviera, at Palermo, Corfu, Cairo; or in islands like Madeira, the Canaries, or those of the Southern Pacific. If the patient's means are ample, this arrangement is the best than can be made for his advantage. [*Vide* art. "Climate in Disease," vol. i. p. 247.]

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PHTHISIS PULMONALIS

CAUSES.—The causation of phthisis pulmonalis is a matter of far-reaching importance to the human race, inasmuch as statistics show that one-seventh of the total death-rate of the world is attributable to this disease.

Phthisis was known to Hippocrates (460-377 B.C.); and in all probability it has existed from the earliest times.

Geography.—Laborious and careful research has established the fact that the geographical distribution of phthisis is coextensive with the habitable regions of the globe. We have the high authority of Hirsch for the statement that pulmonary consumption is "a disease of all times and countries."

But although it cannot be admitted that any part of the world manifests a complete immunity from this disease, Lombard's maps show that it is all but absent in certain Arctic regions, deserts, and places situated at great altitudes; in other words, as pointed out by Dr. Ransome, just where the population is most scanty. If we survey the statistics of various countries, a special incidence on certain districts and towns comes out in the clearest manner. It is perhaps doubtful, as Hirsch remarks, whether a comparison of the statistics of different countries possesses the same value as a study of the returns of individual towns or districts, which ensure a greater degree of accuracy.

Climate.—The influence of climate has been much discussed. Some writers hold that phthisis is commoner in hot than in cold countries; others again consider that it is of more frequent occurrence in temperate climates. But a review of the information at present available leads to the conclusion adopted by Hirsch, that "the mean level of the temperature has no significance for the frequency or rarity of phthisis in any locality." A few examples will illustrate this point. The mortality from this cause in Iceland is very low, whereas in North Greenland phthisis is one of the commonest causes of death. On the north coast of Africa, Morocco and Algiers are distinguished by a remarkable freedom from consumption; but at Tunis, and at Alexandria and Damietta on the sea-coast of Egypt, the disease is very prevalent: in the interior of Upper and Lower Egypt, on the other hand, phthisis is decidedly uncommon. Other discrepancies equally striking might be quoted to prove that places sharing a similar climate may widely differ in their phthisis death-rate.

Consumption seems to follow in the wake of advancing civilisation, especially where men congregate together in large numbers. All accounts agree as to its extraordinary prevalence in New Caledonia, Hawaii, Tahiti, and other South Pacific Islands. There is a strong consensus of opinion that phthisis has become far more rife in these parts, and also among the Maoris of New Zealand, since the date of the European immigration; when, as Hirsch says, "the natives began to adopt the manners and habits of the Europeans." Until recent times the mortality from this disease among the troops of the British Army in the most widely separated parts of the world was deplorably high, more particularly in times of peace; and often considerably exceeded the mortality of the resident population. Improved hygienic arrangements in the barracks, especially as regards overcrowding and ventilation, have reduced the death-rate from phthisis in the most remarkable manner.

Such facts cannot be reconciled with the belief that climate is an important etiological factor. But while the evidence negatives the opinion that hot climates favour phthisis, all authors are agreed that the disease in tropical countries assumes a most acute and virulent type.

Moisture.—A maleficent influence has been ascribed to moisture of the atmosphere and soil. In America, Bowditch was led to believe, by an inquiry into the incidence of phthisis on the inhabitants of certain places, that the disease is most prevalent in areas where the soil is impregnated with moisture. He found in certain localities that efficient drainage was followed by a diminution of phthisis. Dr. Milroy's investigations in Scotland gave similar results. Working out the same idea in England, a few years later, Sir George Buchanan made the discovery that in several towns the phthisis death-rate had undergone a notable decrease since the introduction of an improved system of sewerage—a result which he attributed to draining of the subsoil water. He accordingly expressed the opinion that the mortality from consumption is directly related to the degree of dampness of the soil. But there are certain considerations which make it difficult to regard this conclusion as one of general application. For example, in one of the towns investigated by Buchanan the mortality from phthisis rose remarkably after carrying out the drainage work; moreover, in Berlin, and some other towns in Germany and in England, improvement in drainage has not been followed by the good results anticipated by Buchanan. Lastly, in some of the districts quoted by Buchanan in support of his hypothesis, subsequent investigation by Dr. Kelly has elicited the fact that the diminution in the phthisis rate noted at first has not been sustained in recent years; and, as Dr. Payne remarks, although the subsoil of London is becoming drier every year, owing to the large area covered by houses and almost impervious pavements, there is no corresponding decrease in the amount of phthisis in this town. It is indeed a significant fact that in so wet a country as Holland the death-rate from phthisis is rather low. From these considerations we are driven to admit with Hirsch that other etiological factors beside the influence of soil are probably concerned—factors that "serve to neutralise the benefits even of the most favourable conditions of soil."

Altitude.—Observations made in divers parts of the world have left no doubt of the infrequency of phthisis at higher altitudes, though instances of the disease are not wanting even at the very highest points. The sparseness of the population at such levels may account to some extent for their relative immunity, but not entirely; for in some large commercial and not very sanitary towns in Mexico, and on the Andes, situated at an elevation of from 7000 to 13,000 feet, the extrême rarity of phthisis is generally admitted; a proof, as Hirsch writes, "that the influences which go with very considerable altitudes have the power to overcome those detrimental things that arise from a bad kind of hygiene and social life, in so far as they tend to produce consumption."

Statistics from Switzerland strongly confirm the opinion that a great elevation affords some protection, though they supply no proof of anything like complete immunity. The explanation of the beneficial action of altitude is by no means clear. The extreme freedom of the air from impurities of all kinds and the dryness of the atmosphere have been alleged as the principal causes. But in the case of the unsanitary towns on the Andes above referred to, the absence of organic atmospheric impurities cannot be assumed; and that dryness of the air does not in itself confer any protection is clearly shown by the returns from the towns on the sea-coast of Egypt to which attention has already been directed. Hirsch makes the suggestion that people living at great elevations and breathing rarefied air are obliged to make deeper inspiration, and acquire in consequence a more vigorous development of the respiratory organs, which are thus enabled to offer a more powerful resistance to external influences. The bearing of this view on the bacillary origin of the disease will be discussed further on.

No *race* is exempt from the ravages of consumption. Hirsch states that among the Kanakas—the natives of New Caledonia—two-fifths of the total mortality is due to phthisis. And Dr. Osler states, on the authority of Surgeon Kennedy, that the mortality from this cause in a tribe of Red Indians of the Rocky Mountains living in a splendid climate amounts to 23 per cent of the total death-rate. The Negro appears to be extremely vulnerable, especially when removed from his own country; and in this race phthisis runs a very acute course.

The evidence with regard to the Jews is somewhat ambiguous. There is a general impression that Jews are less afflicted with tuberculosis than Christians. This has been accounted for by the more careful selection of carcases in Jewish slaughter-houses, and by the more frequent house-cleanings practised by the Hebrews. There is some reason to believe that this favourable estimate applies only to the well-to-do members of the community. Further investigation must decide whether the current opinion is correct or not.

Sociological.—We have now to consider another class of etiological factors; the density of population, and certain injurious influences connected with trades and occupations, particularly those which involve an indoor life.

As the general result of statistical inquiry in different countries, it is clearly proved that the mortality from phthisis is lower in the country than in the towns; and that in the case of towns the mortality on the whole increases with the population. The proneness to phthisis manifested by dwellers in towns seems to be largely connected with overcrowding in rooms badly ventilated and lighted. Numerous investigations have attested the high death-rate from phthisis in convents, sisterhoods, military barracks, and above all in prisons. These institutions have been notorious for overcrowding and defective ventilation. In jails and convents insufficient food and indoor confinement have no doubt militated also against the health of the inmates; but these factors cannot be said to have been operative in the case of military barracks. Happily the hygienic reforms of recent times have effected an enormous reduction in the mortality from phthisis in such institutions.

Sedentary occupations, whether in town or country, appear to dispose to the disease. Certain trades, particularly those which are associated with much dust, enjoy an evil distinction on account of the prevalence of consumption among their workers. Attention was chiefly directed to this point by Greenhow's excellent reports, in which he traced the influence of dusty occupations in originating diseases of the lungs.

It is generally believed that the sharper particles of dust are most injurious. Flint workers, needle-polishers, file-cutters, grinders and potters supply the largest contingent of pulmonary diseases. It is still undecided whether most cases of this sort attributable to dust ("pneumonoconioses" of Zenker) are of a tuberculous nature or not. Some authors, among whom is Hilton Fagge, have asserted that practically all these affections of the lungs are tuberculous. Others, including physicians who have seen much of this form of disease, refuse to admit the truth of this statement. It seems to be a fact that in some, perhaps in most of such cases manifest tuberculous lesions are found after death; but even then it may sometimes be difficult to decide whether tuberculosis constitutes the substantive disease, or whether it has implanted itself, as a secondary process, in lungs already the seat of fibroid changes. The researches of Zenker, Virchow, and others leave little doubt as to the occurrence of circumscribed non-tubercular fibroid lesions of the lungs, in consequence of the irritation of finely divided particles of iron and other metallic or mineral substances. But it remains to be proved that massive induration of the lung can be produced by this cause alone without the coexistence of tuberculous disease. This much, however, is certain, that pulmonary affections in persons following dusty occupations, if not always in the first instance tuberculous, are very liable to become so; the chronic inflammation of the bronchi and lung being favourable to the development of this infection.

The comparatively small post-mortem experience in this variety of pulmonary disease which has fallen to my share has almost invariably revealed the presence of chronic tuberculous lesions associated with excessive blackish pigmentation of the lungs. In one or two cases where no evidence of tuberculosis could be detected the lesions consisted of scattered patches of fibrous induration in the peribronchial districts. For a full account of pneumonoconiosis the reader is referred to the article on the subject in the present volume (p. 242).

No *age* is exempt from pulmonary tuberculosis, though it is less common at the two extremes of life.

The view, which prevailed until recent times, that infants and children under two years of age are scarcely ever attacked, has been proved to be incorrect. Landouzy found that in several fatal cases of broncho-pneumonia in children under two years, some of which during life were ascribed to measles or cold, tubercle bacilli were present in the broncho-pneumonic patches; although the naked-eye appearances of tuberculosis were not recognisable. These observations were made at an infant asylum in Paris, in which institution one-third of the deaths among the children proved to be the result of some form of tuberculosis.

Sex.—The following statistical account by Dr. W. Ogle, formerly Registrar-General, deals with the subject of age and sex on a very large scale.

"There is practically no difference between the two sexes in their respective liabilities to death from phthisis when all question of age is put aside. For the mean annual mortality of males on an average of thirty years from this cause is 2418, and of females 2428 per million living. . . . But when instead of taking the aggregate rates, that is, the death-rates of each sex *en bloc*, irrespectively of age, we take the rates at each successive age period, there are found to be very remarkable differences between the sexes. In the first quinquennium of life (0-5) the male and female rates are pretty nearly the same, the male being only very slightly the higher. In the next five age periods, covering between them the interval between the ends of the fifth and thirty-fifth years of life, the female rate is in marked excess of the male rate, the excess being especially notable in the periods from ten to twenty years of age. After the thirty-fifth year the reverse is the case, and the male rate becomes the higher, and remains so in each age period to the end of life.

TABLE A

(Slightly abridged)

Mean Annual Mortality from Phthisis (1851-80) per 1,000,000 living at all ages, and at twelve successive age periods.

	All Ages.	0	5	10	15	20	25	35	45	55	65	75 and upwards.
Both sexes .	2423	1013	461	838	2549	3742	4060	3954	3313	2648	1687	613
Males . .	2418	1034	432	616	2088	3676	3941	4097	3850	3274	2112	730
Females .	2428	993	491	1061	3008	3798	4165	3826	2812	2075	1322	523

TABLE D

Mean Annual Mortality (1861-80) per 1,000,000 living of Children in each Year of the first Quinquennium of Life from Phthisis, Males and Females.

	Under 1	1	2	3	4	All under 5 Years.
Males . . .	1589	1341	634	394	339	880
Females . .	706	1295	655	409	360	842

"It appears from this table that the male death-rate from phthisis in the first year of life is more than twice as high as the female rate; in the second year it is also the higher, but only to the extent of about 3½ per cent; while in the third, fourth, and fifth years of life the female rate is slightly the higher; namely, 3·3 per cent in the third year, 3·8 per cent in the fourth year, and 6·2 per cent in the fifth. These percentage differences are slight; but they run in an ascending series, and, in combination with the figures in Table A, seem to justify the statement that the female liability to phthisis begins to exceed the male liability in the third year of life, and continues to be in excess till somewhere about thirty-five years of age—the maximum of excess being in the ten to twenty years of age period, when the excess reaches 50 per cent."

Dr. Ogle expressly states that the contrast between the male and female mortality described above characterises pulmonary phthisis; and that no similar contrast is observable in the death-rate from other tuberculous diseases.

It is not easy to account for the great excess of the male rate in the first year of life. But as regards the decided excess of the female mortality over the male between the ages of ten and twenty, Dr. J. F. Payne points out that it is at this period of life that the difference between the outdoor life of boys and the indoor life of girls begins; and he suggests that the excessive female mortality at this age is connected with the existence of unfavourable conditions in the house. But, as he further remarks, "the greater liability of the male sex to phthisis after thirty-five years of age seems to be quite unexplained by such considerations."

Dr. Ogle's Tables show that the excess of the female death-rate is not related specially to the child-bearing period, as the excess over the male rate begins to decline after the age of twenty; and after thirty-five the rate has fallen below that of the male sex.

In connection with differences of sex we may now briefly consider the influence of menstruation, pregnancy, parturition and lactation.

Menstruation.—There is no clear evidence that this function, whether at its commencement or subsequently, exerts any definite causative influence. Irregularity or arrest of the catamenia seems at times to be connected with the onset of hæmoptysis; but the nature of this association is probably less intimate than at first sight it appears to be. For it may be safely concluded that a considerable pulmonary hæmorrhage at the outset of the phthisical symptoms is a sure sign that the disease is already of long standing, although perhaps hitherto quite latent. Arrest or disorder of the menstrual flow may occasion reflex vascular disturbance, and so give rise to hæmorrhage from lungs already diseased; but the occurrence is not by any means common.

Pregnancy.—The influence of pregnancy has been much debated. The disease not infrequently appears to extend during this period; but the impossibility of fixing the date at which the disease begins renders the question a difficult one. Wilson Fox considered that the rapid increase of phthisis in woman between the ages of twenty and thirty-five points somewhat strongly to the influence of pregnancy in the development of the disease. The force of this argument is weakened by the fact that the increase in the male rate (see Ogle's Table A) in the same period is almost as pronounced. Dr. R. E. Thompson, as the result of a statistical inquiry into the subject of phthisis in women, concludes "that the susceptibility of single women is rapidly diminished after thirty years of age, while that of married women maintains its intensity between twenty-five and forty years of age (that is, during the child-bearing period)."

Parturition.—It is not common to find that symptoms of phthisis set in directly after parturition, though this event has generally an accelerating effect upon pre-existing disease.

Lactation, by its debilitating influence on weakly women, may no doubt act as an indirect cause. Dr. Pollock states that the periods of puberty, of gestation, of parturition, and of lactation are fraught with danger to persons disposed to phthisis.

The influence of these conditions on the established disease will be considered in a subsequent section.

General depressing influences.—Among the remoter causes of phthisis may be reckoned all conditions that tend to lower the standard of health; such as insufficient food, anxiety, grief, excessive mental work, want of exercise, fresh air, and sunlight. Among diseases that have the same general effect diabetes mellitus must especially be named—a point on which all authorities are now agreed. It is interesting to note the liability of diabetics to another disease of microbic origin, namely, carbuncle.

Concerning the etiological importance of chronic alcoholism, malignant disease, and syphilis, agreement is less general. Both clinical and post-mortem experience alike support the view that topers are prone to tuberculous affections. The frequent association of peritoneal tuberculosis with cirrhosis of the liver is generally recognised by pathologists. It has been urged that alcohol has a preventive action; and that it tends to promote fibroid changes if tuberculosis should be contracted: both statements are at variance with my own experience.

The association of pulmonary tuberculosis and malignant disease of various organs is by no means rare, though it is very uncommon to find evidence of simultaneous activity of the two diseases. Mr. Roger Williams' statistical investigations appear to him to justify the conclusion that the proclivity to cancer is closely allied to the tuberculous diathesis.

A history of syphilis is not rarely obtained from phthisical patients; but if the former disease be possessed of any etiological influence it can only be of an indirect character.

Rheumatism, gout, insanity, chlorosis, dyspepsia have also been regarded as etiological factors; but their connection with phthisis is not intimate.

The frequency with which pulmonary tuberculosis appears after measles, influenza, and, to a less degree, after whooping-cough is well known. Although these diseases cannot be regarded as immediate causes, it must be admitted, more particularly in the case of influenza and measles, that they are apt to precipitate an eruption of tuberculosis; whether it be in consequence of the attendant pyrexia, or of some other action of their specific virus. There is more to be said in favour of the view that a latent tuberculous focus is lighted up, than that tuberculosis is initiated by the presence of another disease. On the other hand, it is possible that influenza and measles may cause certain changes in the bronchial and pulmonary epithelium which result in a lowering of their power of resistance, and thus lay them open to the invasion of tubercle.

Pneumonia.—It is an old belief that croupous pneumonia may terminate in phthisis; but it is now quite certain that this sequence of events is infinitely rare. Two principal fallacies have been concerned in the origination of this erroneous opinion: in the first place, certain rare cases of pulmonary tuberculosis begin with severe constitutional symptoms, and with signs of extensive infiltration of the lung, simulating acute pneumonia; secondly, delayed resolution, or the exceptional supervention of chronic pneumonia, may give the impression that croupous pneumonia has terminated in phthisis. Patients suffering from the latter disease often declare that their illness began with "inflammation of the lungs"; but such statements will seldom stand the test of a critical examination.

Bronchitis not uncommonly appears to have been the starting-point of phthisis, and there is nothing improbable in such a belief; but, more often, careful inquiry will elicit the fact that symptoms of phthisis preceded the attack of bronchitis. In many instances where the tuberculosis appeared to have supervened on chronic bronchitis, an autopsy has demonstrated that the bronchitis was itself symptomatic of chronic pulmonary tuberculosis. The fact that many phthisical persons attribute their illness to a simple catarrh possesses little scientific value; for what ailment do patients not put down to catching cold?

Pleurisy.—The oft-repeated observation that an attack of pleurisy is frequently followed by phthisis, led to the belief that the latter disease is the result of pleurisy. But there is no doubt that, under such circumstances, the original pleurisy has itself been of a tuberculous nature; and secondary, as a rule, to tuberculosis of the lung. There is no reason to think that simple pleurisy disposes to phthisis. In metapneumonic pleurisy, whether sero-fibrinous or purulent, where the effusion is directly due to the pneumonic process, recovery is generally complete and permanent. It is believed by Koch and others that pleuritic adhesions, by impeding the movement of the chest walls and lung, may dispose to phthisis. But patients suffering from deformity of the chest—as the result of kyphoscoliosis, rickets, or caries of the spine—wherein the thoracic movements are greatly restricted—so rarely acquire pulmonary tuberculosis that Rokitansky came to the conclusion that the two affections are antagonistic.

Traumatism has been supposed to play an important part in the causation of phthisis. Mendelssohn has published nine cases of his own, with a reference to seventeen other recorded cases, in which injuries to the chest, of various kinds, were followed by pulmonary tuberculosis. In some instances hæmoptysis occurred at the time of the injury or soon afterwards; in others cough and symptoms of pleurisy ensued within a few days or weeks. In one or two cases an interval of a few months, and, in one case, of two years, separated the accident from the appearance of definite symptoms. Mendelssohn ascribes the occurrence of phthisis in such circumstances to laceration or contusion of the lung, and infiltration of its tissues with blood or inflammatory products, favouring the entrance and germination of the tubercle bacilli, which he assumes to be more or less ubiquitous. From the rapidity with which pulmonary symptoms appeared in most of these instances, it seems more reasonable to suppose that injury to the chest wall may rouse into activity some latent tuberculous focus, possibly by laceration or loosening of its fibrous capsule. As the result of direct questions put to many hundred patients suffering from phthisis, I have met with but a comparatively small number who referred their complaints to an injury of any kind. In one or two instances, when the patient gave a history of injury to one side of the chest, physical signs of disease were confined to the opposite side.

Antagonism of other diseases.—Ague has been said to confer a protection against phthisis, but investigations in malarious localities in various quarters of the world have proved that no such antagonism exists.

An attack of erysipelas has been followed by arrest of the pulmonary disease in a few recorded instances.

Disease of the heart.—Rokitansky taught that all conditions which induce a state of venosity of the blood impart an immunity from tuberculosis. Among the affections included in this category he placed cardiac dilatation and hypertrophy, whether primary or the result of valvular disease, congenital malformation of the heart and great vessels, aneurysms, deformity of the chest depending on rickets, lateral curvature or caries of the spine, pleural effusions, chronic bronchitis, emphysema, bronchial dilatation, pregnancy, or of any other condition tending to obstruct the passage of venous blood through the right side of the heart. The relation of some of these affections to phthisis has been already mentioned; but the influence of cardiac disease must now be considered. It is undoubtedly uncommon to find phthisis and disease of the heart in the same patient, but this association is by no means so rare as Rokitansky's statement would imply. Most writers agree in saying that mitral stenosis is scarcely ever met with in this association: this combination is rare, but I have seen at least a dozen clinical examples; and in five other cases the two conditions were found on post-mortem examination to be associated.

Rokitansky's view that the antagonism of the two affections depends on the venosity of the blood has been objected to by Lebert and others, on the ground that the subjects of congenital stenosis of the pulmonary artery are exceedingly prone to contract tuberculosis. But in such persons, as Lebert himself admits, the lungs are often small and undeveloped, and their nutrition must be below the average. And, although the bronchial arteries are abnormally developed, in order to supplement the pulmonary circulation, the lungs are still very inadequately supplied with blood, and are less capable than normal lungs of resisting the action of the tuberculous virus. It appears then that, in the case of pulmonary stenosis, increased venosity of the blood is not the sole or perhaps the chief influence at work; and Lebert's objection is possibly not so fundamental as it has been held to be. Dr. Pollock considers that hypertrophy and dilatation of the heart retard the progress of tuberculosis, and that under such conditions a prolonged duration may safely be anticipated. This observation is a very true one, and affords support to the theory of antagonism.

It seems, then, that Rokitansky's doctrine is true, though true in a more limited sense than that in which he intended it to be taken. All diseases of the heart which bring about a passive congestion of the lungs, and consequently an increased venosity of the blood, confer a certain degree of protection against pulmonary tuberculosis; but, in the words of Peacock, "this opposition certainly in no degree amounts to an incompatibility."

Gout.—In the rare instances in which gouty persons acquire tuberculosis the disease runs a very chronic course.

Infection.—The doctrine of the infectious nature of tuberculosis, promulgated by Villemin in 1865, was verified by Robert Koch's discovery in 1882 of the immediate cause of the disease, the tubercle bacillus. After much difficulty he succeeded in isolating and cultivating the microbe. In artificial nutrient media the bacillus was found to grow with extreme slowness. Pure cultures inoculated into healthy animals produced tuberculosis with unfailing certainty. From the fact that the micro-organism can only be cultivated within certain narrow limits of temperature (82° to 105° F., the best temperature being that of the interior of the human body), Koch regarded the bacilli as true parasites, "that is, as finding the conditions necessary to their existence only in the animal or human organisms."

These facts have been confirmed by many observers, but Sir H. Beevor, by means of the method of cultivation introduced by Nocard and Roux, claims to have obtained a very slow growth at a temperature of 60° F. If this observation should be confirmed, the tubercle bacillus could no longer be considered to be an obligatory parasite. The experience of all investigators supports Koch's statement that the microbe resists prolonged drying for months; though when exposed to the action of putrefaction it loses its virulence much sooner. The presence of oxygen is necessary for the growth of the bacillus; sunlight has been said to retard or prevent it.

Persons suffering from tuberculous disease of the lungs are constantly expectorating tubercle bacilli in enormous numbers. That the sputa are infectious has been abundantly proved by experiments on animals. Koch and many others consider that tuberculous sputum is the chief source of the parasite. The extreme tenacity of life which characterises this bacillus warns us that the sputum is dangerous long after it has been expelled from the lungs of a phthisical patient. In many well-authenticated cases accidental inoculation of human beings with sputum or other material derived from tuberculous persons has been followed by local or generalised tuberculosis. In one case, a patient dying of gangrene of the leg was inoculated with tuberculous sputum, and at his death three weeks later a few recent tubercles were found in one lung.

Accidental inoculation of the skin, mostly of the hands, has occurred in different ways; for instance, from washing soiled linen of tuberculous people; by a scratch from a broken spittoon used by a phthisical patient; from wearing the earrings of a person that had died of phthisis; by the prick of a morphia syringe; by post-mortem examinations of tuberculous men or animals. In most of these cases the tuberculosis remained localised, and, in some instances, the disease was cured by timely excision of the affected parts. Ritual circumcision in Jewish infants has been followed in several cases by tuberculous ulceration of the prepuce and swelling of the inguinal glands: in some instances it was proved that the operator, himself tuberculous, had sucked the wound to stop the bleeding; in one case where a phthisical operator had not employed suction, he had squirted wine from his mouth over the wound. Bacteriological examination of the ulcer and enlarged glands of the infant, and of the sputum of the operator, was carried out in some instances, and established the infective and tuberculous nature of the process beyond all doubt. But while the inoculability of tuberculous sputum can no longer be denied, it is nevertheless apparent that direct inoculation is a rare occurrence in man, and in no way accounts for the great mass of human tuberculosis. The rarity of this mode of infection is explained by Baumgarten's discovery that tuberculosis cannot be induced by inoculation of the superficial layers of the skin, subcutaneous puncture being required to ensure a successful result.

The fact that the disease, in the great majority of cases, appears to begin in the lungs suggested to Koch that the tubercle bacilli enter the body by the air passages. He further expressed the belief that the bacilli were derived from dried sputum which had become pulverised, diffused in the atmosphere, and inhaled into the lungs. This view has been adopted by most subsequent writers. It may be objected that this mode of infection is insusceptible of direct proof; a striking example, however, has been recorded.

Tappeiner, by spraying tuberculous sputum into a cage where dogs were confined, succeeded in inducing pulmonary tuberculosis in some of the animals. In spite of repeated warnings, Tappeiner's servant, a very robust man aged forty, and free from hereditary taint, insisted on going into the cage, and contracted acute pulmonary tuberculosis from which he died in fourteen weeks. It may be freely admitted that the conditions in this case were not strictly parallel to those that obtain under ordinary circumstances where the amount of tuberculous dust inhaled must be very small; yet the case demonstrates the possibility of man acquiring tuberculosis by inhalation. All observers have admitted the difficulty with which tuberculosis can be communicated to animals by means of inhalation, a fact which Baumgarten maintains is opposed to Koch's conclusions. After the discovery of the tubercle bacillus the view was very generally expressed that the parasite is ubiquitous, and that every one, especially in towns, must be frequently inhaling the microbe. But a careful and extensive research, conducted in Berlin by Cornet, proved that the bacillus is not so widely distributed as had been assumed. The plan which Cornet adopted was to collect dust with sterilised instruments from the walls of hospitals, prisons, asylums, and private houses, and from the public streets. The dust was mixed with sterilised broth and injected, with full antiseptic precautions, into the peritoneal cavity of guinea-pigs. Many of the animals died rapidly of septic peritonitis; others remained in good health, and a certain number contracted tuberculosis. The specimens of dust which communicated tuberculosis to the animals were obtained from private rooms or wards that had been inhabited by phthisical persons; whereas in surgical wards, out-patient departments, and in quarters not occupied by such persons, the dust, as regards tuberculosis, gave negative results.

In the course of experiments made, after Cornet's method, by Dr. Heron and Dr. Chaplin with dust from the Victoria Park Chest Hospital, only two out of a total of a hundred guinea-pigs inoculated were attacked by tuberculosis. In both these cases the particular specimen of dust came from the main ventilating shaft, which had not been swept for forty years. Dust taken from the wards, out-patient waiting-room, and pathological laboratory failed to cause tuberculous infection in a single instance.

Dr. C. T. Williams succeeded in detecting a few tubercle bacilli in the air of the Brompton Hospital. His method consisted in the exposure of glass plates, smeared with glycerine, in the ventilating shafts of a ward set apart for phthisical patients. After some days the plates were examined microscopically and a few bacilli were found. It is very probable that in this case the microbes were conveyed to the glass plates with dust.

Dr. Ransome, by condensing the breath of consumptive people in a glass globe surrounded with a freezing mixture, was able to discover a few tubercle bacilli in two cases. But in such experiments it must be difficult to prevent patients from coughing and expectorating minute quantities of sputum or saliva which may contain bacilli. Numerous workers have failed to verify these observations, and we may assume that the bacillus is not exhaled from the lungs during ordinary respiration. Cornet believes that the great majority of cases of pulmonary tuberculosis are the result of inhalation of dried sputum in association with dust.

Others think that the alimentary canal is a more important channel of infection. Experiments on animals have proved that tuberculous material when swallowed may induce tuberculosis of the mesenteric glands and intestine. Considering that tubercle bacilli may lie dormant amid the dust of houses inhabited by phthisical people, it is not improbable that children may accidentally contaminate their food and thus acquire abdominal tuberculosis. Milk and butter from tuberculous cows, and the flesh of oxen, pigs, and fowls, when imperfectly cooked, may also communicate the disease.

For a detailed discussion of this topic the reader is referred to the article "Tuberculosis" (vol. ii. p. 29). The preponderance of primary disease of the lungs, however, seems to support Koch's view that the virus in most instances enters the body through the respiratory system.

The difficulty found in producing tuberculosis in animals by inhalation may seem to oppose this view. But the pulmonary affection in man, as compared with the artificially induced disease in animals like guinea-pigs and rabbits, is always a chronic process. If an animal after inoculation fail to show evidence of disease in a few weeks or months, the experiment is generally regarded as unsuccessful; whereas, to make the parallel complete, continued observation for a much longer period would be required. For in man the period of latency of pulmonary tuberculosis is generally one of months, or even perhaps of years. The success of infection is largely a matter of dose; a very small dose producing a chronic affection or no result at all, a large dose, on the other hand, causing an acute infection. Moreover, variations in the virulence of the bacillus may be evidenced by corresponding differences in the type of disease. Bacilli subjected to the action of desiccation for months are less capable of active growth than when freshly removed from the animal body or from artificial cultivations.

That Cornet's injections of dried tuberculous dust proved fatal to guinea-pigs in a comparatively few weeks does not constitute a serious objection to this conclusion; injections of such dried matter under the skin or into the peritoneal cavity is a much stronger measure than its introduction by inhalation; for in the latter case the ciliary movement of the epithelium and the vital resistance of the cells of the respiratory tract represent a powerful defensive mechanism that cannot be claimed for the subcutaneous tissue or the serous membranes. The existence of such a mechanism is well shown by the history of anthracosis and other dust affections of the lungs. Children and animals living in the dusty atmosphere of towns, as Baumgarten remarks, seldom show any of the pigmentation of the lungs and bronchial glands, which is never altogether absent in adults living under similar conditions. It seems as if the ciliary action of the epithelial cells can remove all the foreign particles introduced with the air up to a certain point and for a certain length of time; but after a time the carbon and other particles enter the lymphatic vessels and become deposited in the lungs and in the neighbouring bronchial glands.

It is very probable that tubercle bacilli entering the mouth are taken up by the tonsils and carried to the cervical glands; thence they may pass into the large lymphatic vessels, and thus ultimately reach the lungs.

House infection.—A considerable number of observations have now been recorded in support of the view that the tuberculous virus clings to certain dwellings.

Dr. Ransome's investigations in Manchester and Salford have shown that tuberculosis is especially apt to haunt houses situated in close courts, narrow streets, and, above all, houses built back to back, where ventilation is necessarily defective. Similar observations have been made in America and Germany. In some of the cases published the evidence is very strong, as, for instance, in the following by Engelmann. A newly-built flat, in a fairly sanitary condition, but badly lighted and ventilated, had been occupied for eight years by three families in succession; all of them had presented a clean bill of health until the family X took up their residence in the same quarters. In this family the mother was consumptive when she came, and died in the flat. Shortly afterwards the family left, having lived there for one year only. The flat was next occupied by the family Y, of seven persons, all healthy; after a year's stay they left, and some years later the father, mother, and one son died of phthisis, and a boy of chronic peritonitis. A third family, Z, all healthy to begin with, next took the rooms: one child died of meningitis, another of marasmus, and a third contracted hip disease: subsequently the father died of phthisis, another child of meningitis, the mother acquired consumption, and a child became scrofulous. A fourth healthy family, W, next came into residence; after a time the mother became phthisical, and two children died of meningitis. In reference to these facts, Dr. Payne remarks: "Summing up the history it appears that for eight years the dwelling was free from tuberculous diseases. Then came one year's tenancy by a person already tuberculous. After this, in a period of twelve years, at least twelve cases of tuberculous disease were traced to this source. It is noted that the dwelling was never vacant, the new tenants entering while it was, so to speak, still warm from the last; and during the whole period it was never painted or cleaned." In other parts of the same house, where cleaning was not neglected, but the conditions were otherwise the same, no cases of tuberculosis could be traced. The facts point strongly to infection in the case of the third and fourth families (Z and W); but in respect to the second family (Y) the evidence is not so convincing, as according to Engelmann's statement some years elapsed between the tenancy of the infected house and the deaths of some of the members from tuberculosis. The hypothesis that the virus is air-borne, and intimately connected with dust, helps us to understand how house-infection may come about. In most of the instances recorded the victims lived in small, ill-ventilated rooms, so that the chances of infection were thereby much increased. The smaller the room the less the likelihood of adequate ventilation, and the greater the opportunity for the accumulation of dust.

An important side light is thrown on this part of the subject by the returns of the mortality from phthisis in the male and female sex among certain agricultural populations in England and Germany, which show a marked excess of the female over the male death-rate. In other words, the males who lead an outdoor life suffer much less from consumption than the females, who spend most of their time indoors. Although, as Payne points out, this disparity in the phthisis death-rate may be explained on the ground that the open-air life of the men is healthier, it is quite as logical to say that the indoor life of the women exposes them to some injurious influence derived from the dwellings. We know that the tubercle bacillus is apt to cling to ill-ventilated and insufficiently cleaned rooms inhabited by phthisical persons, conditions only too well fulfilled in the houses of the poor. It is hard to resist the conviction that these facts are most readily to be explained by the more prolonged exposure of the women to the risk of house infection. In towns the male death-rate from phthisis exceeds the female. The difference here, no doubt, depends on the unfavourable conditions under which men commonly work in rooms badly ventilated and dusty.

The great preponderance of the phthisis rate on the female side between the ages of ten and twenty, as shown by Ogle's tables, corresponding, as it does, with the period in which the outdoor life of boys and the indoor life of girls differ most widely, points in no uncertain manner to the dwellings as the source of the mischief.

Contagion.—The contagious nature of phthisis, long an article of popular belief in parts of Southern Europe, appears then to derive confirmation from Koch's discovery.

Since 1882 many cases have been published in support of this doctrine. In most of these the parties concerned were married couples; and the disease seems to have been communicated from husband to wife more frequently than in the reverse direction. We may briefly consider the ways in which contagion may possibly occur.

(i.) By the skin. Direct inoculation has been already discussed and shown to be a very exceptional occurrence.

(ii.) By the alimentary canal. Tubercle bacilli might be accidentally introduced into the mouth, as by kissing; or less directly, by the use of knives, forks, spoons, or drinking-vessels. The great rarity of primary disease of the tongue, oral cavity, and alimentary tract generally, and the comparative infrequency of primary tuberculosis of the mesenteric glands, except in children, negative this mode of infection.

(iii.) By the respiratory system. Although the bacilli are not given off in the breath, the possibility of their being expelled by coughing with small quantities of mucus or saliva must be admitted; but this accident cannot be regarded as playing an important part.

(iv.) By the generative system. Where the generative organs are tuberculous, it is possible that contagion may take place during sexual intercourse, in either direction; but the occurrence of primary tuberculosis of these organs in either sex is extremely rare. Direct contagion must be very uncommon, and it can have little bearing on the causation of the disease. It is possible that the bacillus or its spores may pass with the sperm cell to the ovum without infecting the mother, as is believed to occur in syphilis. This question will be again referred to under the head of heredity.

If the views already expressed as to the part played by dried sputum and tuberculous dust be correct, there is no necessity to invoke the supposition of direct contagion, which, in truth, stands on no firm foundations. Husband and wife living in the closest relationship and in the same rooms, are necessarily exposed to the same risks; although the member who spends most time in the infected rooms is more likely to contract the disease. If husband or wife be already tuberculous a fresh dwelling may be converted into a focus of infection, and the healthy one may indirectly acquire phthisis by inhaling tuberculous dust.

Heredity.—Phthisis has always been accounted one of the most hereditary of all diseases. Numerous statistics, dealing with this point, are at hand; but, seeing that some refer to parental inheritance only, while others include collateral influence also, and in view of the fact that information concerning collaterals is less likely to be precise, we may confine our attention more particularly to parental inheritance.

The extent to which parental heredity is manifested in the subjects of phthisis has been very variously stated, Portal rating it as high as 66 per cent, Louis as low as 10 per cent. We may, perhaps, regard 30 per cent as about the proportion in which, according to most investigators, a history of parental heredity can be obtained. It has been maintained that fathers transmit to sons more frequently than to daughters, mothers to daughters more frequently than to sons. But the statistics of Walshe, R. E. Thompson, and Wilson Fox do not support this assertion. Heredity is generally but not universally regarded as playing a more important part in females than in males. It is stated that more female than male patients give a history of phthisis in the parent; and that among all hereditary cases maternal is in excess of paternal inheritance. In some cases inheritance seems to have been derived from grandparents or great-grandparents, the parents having played the part of silent carriers of the disease. According to several observers, phthisis is manifested at an earlier age in those that evince an hereditary taint. After the age of twenty-five the acquired cases equal the inherited, and ultimately out-number them. According to Dr. R. E. Thompson, a greater severity of form and a shorter duration of life characterise the hereditary cases; but the experience of Dr. C. T. Williams does not confirm this conclusion.

Enough has now been said to show that, after all the labour expended on this subject, no general agreement has yet been reached.

It is evident that the investigation of this question is exposed to many fallacies, a few of which may be mentioned. In the first place, many deaths of parents and grand-parents may have been wrongly attributed to bronchitis, pleurisy, or pneumonia when the affection was really tuberculous. Against this, of course, in other cases death may have been erroneously ascribed to tuberculous disease. In dealing with a large number of cases these opposing fallacies will to some extent neutralise each other. A more important source of error depends on the undoubted fact that many ancestors reputed healthy have been the subject of arrested tuberculosis. Again, parents may not manifest signs of phthisis till after the death of some of their offspring from this cause.

In the case of heredity among collaterals—brothers and sisters, uncles and aunts, cousins—the same fallacies must arise, but with an important addition. In all families, but especially among the poor, the mortality of infants and young children is very high; and there can be no doubt that the existence of tuberculosis at this age is very largely overlooked, death being ascribed to marasmus, diarrhœa, bronchitis, or broncho-pneumonia. On the whole, it seems that the tendency of the fallacies referred to would be to underestimate rather than to exaggerate the influence of heredity. The heredity of phthisis has received two widely different explanations. According to the prevailing opinion, it is not the disease itself that is inherited, but a disposition or tendency to acquire the disease when exposed to the necessary influences; the other view is, that the germ of the disease is directly communicated from the parent to the embryo.

The doctrine of hereditary predisposition has been assailed on more than one ground. In the first place, the existence of a peculiar bodily conformation in the children of phthisical families, the tuberculous and scrofulous diatheses so much insisted upon by some writers, has been called in question. It is admitted that some of the features described are often seen in persons suffering from phthisis, though it is believed that to a considerable extent they are attributable to wasting of the muscles and adipose tissue, or to enlargement of external lymphatic glands, and are, therefore, manifestations of existing disease. These objections seem to be justified; but the hypothesis of hereditary proclivity does not necessitate the assumption of a special bodily habit, and the abandonment of this postulate does not materially weaken the position of those who hold to the doctrine of predisposition. It has been objected that the percentage of family inheritance reckoned up from phthisical patients does not truly represent the influence of heredity, and that the percentage should be compared with the incidence of the disease in healthy families. Moreover, it is suggested that what is inherited is not a special disposition to tuberculosis only, but a general delicacy or vulnerability to adverse conditions of all kinds. According to Beneke this vulnerability is connected with the relatively small size of the heart in such persons. Others again would explain the prevalence of the disease in certain families by the greater opportunities of infection that exist in the dwellings of such persons. The first objection may be admitted as valid; but in order to arrive at accurate conclusions on this basis the subject would require investigation on a much larger scale than has been hitherto attempted. As Dr. Kingston Fowler points out, it is obviously misleading to work back from the consumptive member of a family to the parents, and to deduce the influence of heredity from a comparison of the percentage incidence of phthisis in the children of the phthisical and non-phthisical—a method adopted by some investigators in this field. For this practically assumes that there is a consumptive in every family, and takes no account of the families in which, in many unselected series, no member is phthisical.

With regard to the explanation of heredity on the hypothesis of family infection, it seems that although this may account for many cases it will not explain all. Instances are not wanting where several members of a family, widely separated from one another, have manifested the disease in succession. If, however, the extreme latency of the tubercle bacillus postulated by some writers could be substantiated, the question of heredity would at once assume a new aspect altogether. Before proceeding to discuss the doctrine based upon this hypothesis, it may be pointed out that the existence of a family susceptibility to other infectious diseases—as to typhoid fever, scarlatina, and diphtheria—has long been recognised by epidemiologists.

To Cohnheim we owe the suggestion that heredity depends upon the direct transmission of the tuberculous virus to the embryo—a view which has been further developed by Baumgarten. This author holds that infection of the respiratory and digestive tracts will only account for a small proportion of the cases of tuberculosis; and by a process of exclusion he is led to the belief that heredity is the most potent factor in the continued existence of the disease. After rejecting the notion of hereditary predisposition mainly on the strength of arguments derived from the results of the experimental inoculation of animals, Baumgarten embraces the doctrine of the direct inheritance of the tubercle bacillus or its spores. According to his view the microbe may either be introduced through the placenta and thence infect the fœtus through the umbilical vein ("placental infection"), or it may find access to the ovum itself either in the ovary or after its passage into the Fallopian tube ("germinative or conceptional infection"). In the latter case the microbe would mostly be conveyed by means of spermatozoa, though an observation of Jani's suggests that the bacilli may enter the Fallopian tube from the peritoneal cavity. The possibility of germinative infection from paternal sources cannot be denied in view of the discovery, by Jani and Weigert, of tubercle bacilli in the healthy testes and prostate glands of phthisical men. Virchow objects to the view that germinative infection plays an important rôle in heredity, on the ground that the presence of the bacillus must interfere with or arrest the development of the ovum; but Baumgarten urges that this argument is negatived by the history of congenital syphilis, and by the analogy of the pébrine disease of silkworms. In the case of syphilis, although miscarriages may occur, it commonly happens that the child is apparently healthy at birth, and signs of the disease do not appear for some weeks; a period of latency, therefore, undoubtedly ensues between infection of the ovum or fœtus and the first few weeks of extra-uterine life. In the pébrine disease, which is caused by a psorospermial organism, Pasteur has shown that the ova of the silkworm become infested with the spores of the parasite; but in spite of this the eggs are hatched normally, though the caterpillars ultimately succumb to the growth of the parasite in their bodies.

Baumgarten would explain the latency of the pébrine disease, congenital syphilis, and inherited tuberculosis by the supposition that the actively growing embryonic cells inhibit the development of the respective microbes.

Some interesting researches by Maffucci have an important bearing on this question. Tubercle bacilli from a tuberculous fowl were introduced into fertilised hen's eggs and incubation was allowed to proceed. Maffucci found that the bacilli did not multiply, but underwent a regressive change into granules exhibiting the staining reactions characteristic of the normal bacilli. The chick was hatched out in the usual way, but after about the twentieth day the bacilli began to develop, and a typical tuberculous infection ensued, the liver being conspicuously involved. If the dose of the bacilli introduced be small no visible tubercles form, but the chicken nevertheless dies of extreme marasmus and bacilli are found in the organs in small numbers. The analogy suggested with congenital syphilis, the pébrine disease, and congenital tuberculosis of fowls is both interesting and instructive.

Placental or germinative infection may explain the rare cases in which tuberculosis is found in the fœtus or new-born infants; and also perhaps the less uncommon instances where the disease arises during the first few months of life. But there seems to be no sufficient reason for the belief that the tubercle bacillus or its spores may remain dormant from the time of conception of the ovum to adult or middle life. Baumgarten would go even farther, for he applies his hypothesis to explain atavism occurring in tuberculous families; and would trace the inheritance of the microbe to a grand-parent or even more remote ancestor, when the parents have remained healthy. The evidence in favour of

Baumgarten's hypothesis is not strong, and is mainly drawn from observations on animals. Fœtal tuberculosis has now been demonstrated in several cases in calves, but in man such an occurrence is extremely rare. Landouzy and Martin have published a case where the apparently healthy fœtus of a phthisical mother proved capable of infecting animals with tuberculosis, to show that tubercle bacilli may be present in the tissues without exciting any manifest lesion. The hypothesis of direct inheritance does not appear to be reconcilable with the facts disclosed by Ogle's statistics. A reference to his table shows that the mortality from phthisis declines greatly after the completion of the second year until the tenth year, when it begins to rise again, attaining its maximum from twenty-five to thirty, but maintaining a high level up to the age of sixty-five. Moreover the marked difference in the incidence of the disease on the two sexes between the ages of ten and twenty is quite inexplicable on Baumgarten's theory.

The only conclusion at present warranted is that direct inheritance is of decidedly subordinate importance to extra-uterine infection, however acquired.

PATHOLOGICAL ANATOMY.—Tuberculosis is in its origin a local disease depending on the lodgment and growth of the tubercle bacillus; but in virtue of its infective character it not only extends by continuity from the primary lesion, but it tends also to invade other parts of the body.

Fever and other constitutional effects of tuberculosis are often out of all proportion to the extent of the local disease, and must be ascribed to the circulation in the blood of some as yet unrecognised chemical poison produced by the bacillus.

We have now to consider the changes in the lungs that result from the *invasion* of the tubercle bacillus. The initial lesions exhibit certain differences according to the manner in which the microbe is introduced into the organ. Excluding the comparatively few cases in which the pulmonary disease is due to direct extension from neighbouring lymphatic glands, or from the osseous parietes of the thorax, it may be said that the bacillus gains entrance in one of two ways, through the blood-vessels or through the bronchial tubes. In the former case the entry of a large number of bacilli into the circulating blood gives rise to an eruption of miliary nodules disseminated through the whole lung, and through many organs of the body. In such cases, as was first pointed out by Buhl, a caseous focus will almost invariably be found in some lymphatic gland; or, possibly, in the lung itself. It is probable that the introduction of a small dose of the bacilli may have as its result a circumscribed lesion of the lung. In either case infection is brought about by an embolic process, the microbe being arrested in the alveolar capillaries. The presence of the bacilli in the first instance provokes a specific cellular growth in the capillary wall, but the process soon extends into the cavity of the air sacs, where a similar cell growth develops. If the microbes enter the lung through the air-passages they appear to become arrested in the terminal bronchioles or alveoli, in which parts the epithelium is not ciliated. From the bronchiole the cell growth invades the peribronchial sheath and alveolar cavities, the result being an islet of peribronchitis and broncho-pneumonia. Tuberculous growths, wherever situated, are devoid of blood-vessels. In generalised miliary tuberculosis the pulmonary changes are but a part of a general infection of the body, though the lung may suffer most. As death results in a few weeks at the latest the tubercles in the lungs have not time to go through the usual cycle of changes manifested in the cases which run a more chronic course.

Inasmuch as the lesions of chronic tuberculosis differ in degree rather than in kind it will be convenient to study the process in the chronic form. We have seen that in primary tuberculosis of the lung the bacilli are probably introduced as dust with the air. Since the time of Louis the preference of tuberculosis for the apex of the lung has been universally recognised; the earliest lesions are found about one to two inches below the extreme apex. In rare instances the disease begins in other parts of the lung, as at the base of the lower lobe; but in adults a primary basic origin is exceedingly rare, and is probably not found in more than one in 400 or 500 cases: in children it is relatively less infrequent, but this is due to the fact that in them primary tuberculosis of the bronchial glands is more common and attains to greater proportions than in adults. Many cases of tuberculosis in children apparently basic in origin are really due to direct extension from caseous bronchial glands. In rare cases of irregular localisation, whether in children or adults, the disease has originated in the vertebræ. The special proclivity of the apex of the lung to tuberculosis has been variously explained: this part of the lung undoubtedly possesses a smaller range of movement than the lower portions in consequence of the greater rigidity of the upper ribs, and this condition must favour the retention of foreign matter in the bronchial tubes and alveoli, and will thus favour the lodgment of the bacilli. Moreover, Dr. R. E. Thompson points out that the comparatively fixed position of this part of the thorax tends to keep the bronchial tubes of the apex widely open, whereby the entrance of dust and other extraneous matter is promoted. But in generalised miliary tuberculosis also, where the bacilli enter the lung through the blood-vessels, the lesions are often most advanced at the apex, a fact which points to the existence of a special vulnerability of this part of the lung itself. It has been stated that the circulation in the apex is less vigorous than in other parts of the lung, and that this part of the lung being drier is more susceptible, but of these opinions there is little direct proof. At an early stage of the disease the lesion will be found to consist of one or more small grayish nodules, the centre of which corresponds to a bronchiole; as these nodules increase in size they tend to acquire a racemose shape owing to the growth of miliary granulations at the periphery. In man it is not easy, as a rule, to trace the earliest steps of the process in the primary nodule, as before the patient's death regressive changes have already set in; but from a study of the secondary nodules developed in similar cases we may conclude, as Rindfleisch has long taught, that the process begins in a terminal bronchus and thence spreads to the corresponding lobule—that is to say, the lesion is essentially broncho-pneumonic. In the early stages we find the mucous membrane of the bronchiole swollen and infiltrated with cells, and the surface more or less denuded of epithelium. The cavity of the tube contains mucous secretion mixed with pus cells. Tubercle bacilli may sometimes be recognised in the secretion as well as

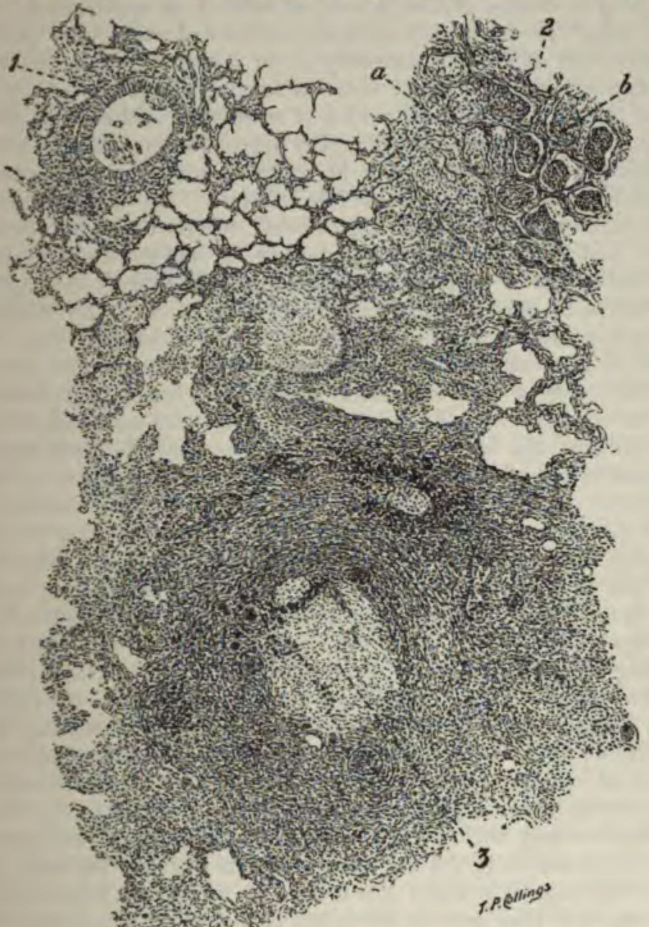

Fig. 6.—Composite photograph taken from three different sections, illustrating the development of the tubercular process. (Low power.) 1. Islet of tubercular peribronchitis, a bronchiole with surrounding tubercular infiltration. 2. Patch of tubercular "pneumonia": *a*, Early stage—alveoli stuffed with large pale epithelioid cells (not recognisable with so low a power); *b*, Later stage—alveoli contain granular necrotic masses, the outlines of the cells having disappeared. 3. Fibro-caseous tubercular nodule, showing a pale amorphous caseous centre embedded in concentrically arranged fibro-cellular connective tissue, which contains a few giant cells in its inner zone. Coarse black carbon particles in places.

in the cellular infiltration. A similar cell growth, with a varying number of bacilli, is found in the peribronchial sheath and in the corresponding alveoli. The tuberculous growth, whether in the bronchiole or in the air-sacs, is seen to consist at first mainly of cells of an epithelial type—epithelioid cells. In some cases the nodule seems to be composed exclusively of small round cells. Subsequently large multinucleated cells ("giant cells") appear singly here and there in varying numbers. At the periphery of the tuberculous area a zone of small lymphoid cell can generally be recognised. In the last-named region the cells are entangled in a scanty meshwork of delicate fibres; but in no cases, save the most chronic, is fibrillation visible at the centre of the tubercle. By degrees the cellular growth extends to neighbouring lobules, where the same process is enacted; and thus the original nodule may become more or less lost in a diffuse infiltration or tuberculous pneumonia. The direct propagation of the disease is brought about by the spread of the tubercle bacilli along the lymph spaces and vessels in the interstitial tissue of the lung. In the course of the tuberculous process the walls of the alveoli and, in a lesser degree, the coats of the small arteries and veins become involved in the cell growth. Perforation of the wall of a pulmonary vein and entrance of the bacilli into the blood is, as was first shown by Weigert, one of the commonest modes of general infection of the body in cases of pulmonary tuberculosis. A small artery may be affected in like manner, and a localised eruption of miliary tubercles take place in the lung in the area of distribution of the affected vessel; but in most cases extra-alveolar lesions are of quite subordinate importance to the changes occurring within the air-sacs. In the most chronic varieties of tuberculosis the *development of fibrous tissue* is the predominant feature; and this leads to a thickening of the interalveolar, peribronchial, interlobular and subpleural connective tissue. *Tubercle bacilli* are found in and among the epithelioid cells, in the giant cells, and occasionally in the small-celled area; but not in the fibrous tissue itself. Except in miliary tuberculosis, and in certain instances of acutely spreading disease, the number of the microbes that can be demonstrated is generally small, and contrasts strongly with the large numbers met with in the artificial tuberculosis of animals. The paucity of the bacilli in the former case agrees with the far more chronic course of the disease in man; but it may be, as Ehrlich suggests, that in certain stages of their growth the bacilli cannot all be successfully stained by our present methods.

The destiny of the tuberculous growth is twofold. In the first place, the cells undergo *necrosis;* their outline becomes indistinct, the nucleus disappears, and the cell is converted into a finely granular or hyaline mass, which then becomes fused with neighbouring cells in a similar state of degeneration. As a result of this change large areas of the affected organ become transformed into opaque, yellowish white material, resembling cheese—a result known as caseous degeneration, or necrosis. The cause of this change is uncertain, but it may depend, as Professor Watson Cheyne suggests, on some chemical poison elaborated by the bacilli. Caseation is regarded by Weigert as an instance of coagulative necrosis. Cheesy foci may remain unaltered for a considerable time; but they are very liable to undergo liquefaction, and when a communication is established with a bronchus the softened material is evacuated and a cavity or vomica is formed. In some cases the process of softening and excavation originates in a small bronchus. Tubercle bacilli are found in immense numbers in the cavities, but in caseous material they are generally very scanty. Cavities also contain various pyogenetic cocci; but, according to some observers, suppuration may be excited not only by such cocci, but also by the tubercle bacillus. A caseous or necrotic change is probably never altogether absent at some period in the development of any tuberculous formation, and, as a rule, this is the prevailing feature; but at the same time, in most cases of pulmonary tuberculosis in man, and in all chronic forms without exception, another process of a conservative or reparative character is recognisable in the shape of a *growth of connective tissue.* This change begins at the margin of the tuberculous area, where it constitutes a species of fibrous capsule. It is doubtful whether this be a direct result of the tuberculous process, or whether it be attributable to reactive inflammation around the tubercle. In some cases the central caseous mass becomes thus shut off from the surrounding parts and complete arrest of the disease is effected. The cheesy matter may subsequently become calcified, or it may be gradually permeated by connective tissue and converted into a solid fibrous knot. Fibrosis and encapsulation are the natural mode of healing. The well-attested frequency with which old fibrous tubercles or calcareous nodules are found in the lungs at post-mortem examinations of patients dying of other diseases, shows that recovery from tuberculosis is by no means so rare as was formerly supposed. In the great majority of cases, however, the fibrous change is more limited, indications of a capsule can scarcely be recognised, and fresh islets of tuberculous disease spring up on the confines of the original patch. These secondary foci, which depend on the spread of the bacilli along the lymphatic spaces, go through similar stages of cell growth, necrosis, and connective tissue development. According as the necrotic or fibrous change preponderates, the case assumes an acute or chronic complexion; but the combinations of the two processes are subject to infinite variety. Thus a case originally of a marked fibroid type may be complicated by the occurrence of acute destructive disease in other parts of the lung; or, again, though this is far less common, rapidly progressing tuberculosis may undergo partial arrest and pass into a chronic fibroid stage. In ordinary cases we find some indications of healing at the apex, while in the more recent lesions necrosis is the predominant factor.

Histogenesis.—The origin of the cells constituting the tubercle has long been a matter of dispute; some authors maintain that they are the result of the proliferation of fixed tissue cells, others regard them as leucocytes that have escaped from the vessels.

An important experimental research by Baumgarten has gone far to prove that the specific tubercle cells are epithelioid in type, and are the offspring of the epithelial, endothelial, and connective tissue cells of the body. In miliary tuberculosis of the lung the process begins with swelling and nuclear division of the endothelium of the alveolar capillaries, of the epithelial cells lining the alveoli and bronchioles, and, in a minor degree, of the interstitial connective tissue cells. For a fuller consideration of this point and for further histological details the reader is referred to the article "Tuberculosis" (vol ii. p. 6). Although in the case of miliary tuberculosis the bacilli reach the lung through the blood-stream and become arrested in the capillaries, yet the earliest lesions consist predominantly of an accumulation of epithelioid cells within the air-sacs; and miliary tuberculosis of the lungs has not inaptly been designated a miliary pneumonia.

For simplicity's sake the development of the disease has been sketched as it affects individual sections of the lung; but as a matter of fact the process is far more complicated. While separate foci of tubercle go through the stages just described, various *secondary changes* of a congestive, inflammatory, and œdematous nature commonly ensue in the intervening portions of lung. These conditions are partly the result of compensatory hyperæmia; but in the main they depend on obstruction of the smaller bronchi and on the lobular collapse that follows.

Collapse soon passes into *broncho-pneumonia*, which, if not from the first actually tuberculous, soon becomes so. In such cases scattered caseous spots are seen embedded in reddish, solidified lung. The pneumonic condition, at first, is usually patchy or lobular in distribution; though from the coalition of numerous individual foci the consolidation may ultimately involve the greater part of one lobe. On section the surface is moist, flat, and glazed; seldom dry and granular, as in croupous pneumonia: this difference depends on the fact that the exudation is mainly composed of cells and œdematous fluid, and contains little fibrin.

In some instances the consolidation has a pale pinkish gray gelatinous appearance, the "gelatiniform infiltration" of Laennec. This condition seems to be due to the existence of marked anæmia and œdema in addition to the alveolar catarrh and collapse. In such infiltrations it is not uncommon to discover small specks of caseous necrosis, which stamp the process as essentially tuberculous. Sometimes a large area or even a whole lobe presents a more or less uniform grayish or yellowish consolidation known as caseous pneumonia. In such cases, as a rule, old caseous foci, or a cavity, will be found at the apex, suggesting the secondary nature of the diffuse infiltration.

Acute croupous or lobar pneumonia is stated by many authors to be a common termination of pulmonary tuberculosis. I am convinced that this is an error. An experience of some thousand necropsies of cases of this disease has not furnished me with more than one undoubted instance in which progressive tuberculosis of the lung was complicated by acute fibrinous pneumonia. During the first influenza epidemic, two or three cases, in patients who succumbed to an acute pneumonia of more or less lobar dimensions, revealed an œdematous, ill-defined consolidation, consisting microscopically of cells and œdematous fluid without any fibrin; these may have been modified instances of acute lobar pneumonia, but with these few exceptions the above statement holds good.

Phthisical patients are indeed often cut off by acute intercurrent disease of the lower lobes; but this is essentially broncho-pneumonic, and probably depends on the inhalation of septic microbes from ulcerative cavities in the lung.

In the more chronic cases a localised *emphysema* is not uncommon, especially where contracting lesions are separated by tracts of unaltered lung. This condition, which is most pronounced towards the apex and anterior margin of the upper lobe, may be so extensive as almost to mask the original disease. The affected lung may present the appearance of large superficial bullæ, or the form of emphysema may be more diffuse. The surface of such portions is often puckered from the contraction of subjacent fibrous patches or cavities. Emphysema in these circumstances is compensatory, and results from obliteration of adjacent alveoli. It is necessary to distinguish clearly between true emphysema, which is a degenerative atrophy of the alveolar walls and capillaries, and what may best be described as pulmonary distension. When one lung is contracted, the opposite lung undergoes vicarious enlargement, the alveoli becoming uniformly enlarged without being otherwise altered; the expanding lung passes across the middle line of the sternum and encroaches upon the space formerly occupied by its fellow. The effect of this enlargement is an increase of alveolar surface, and consequently an improved aeration of the blood. This condition has been named "hypertrophy of the lung," a description which implies increased function, and is therefore strictly correct. It is possible, however, that this condition may, in time, pass into true emphysema.

Cylindrical *dilatation of the smaller bronchi* is not uncommon, and may be found in any part of the lung, whether its texture be spongy or indurated. Bronchiectasis is to be attributed mainly to the positive expiratory pressure of cough acting on the bronchial walls softened by inflammatory or other changes. The existence of contracting fibrous tissue in the surrounding lung, by drawing apart the walls of the bronchi, will further contribute to the dilatation.

In all chronic cases *pigmentation* is a more or less marked feature; it depends mainly on the deposit of particles of carbon derived from the atmosphere. Old fibroid lesions have a blackish or slaty colour, which contrasts sharply with the red, yellow, or grayish tint of other parts of the lung.

The process of softening of the caseous material and the formation of cavities have been already briefly alluded to. The liquefaction which occurs has been likened by Duclaux to the ripening of cheese; but the nature of the chemical transformation is still unknown. The shape of *pulmonary cavities* varies greatly. They may be rounded or oval; but more often they are sinuous or anfractuous, in consequence of the coalescence of separate vomicæ, and of the irregular extension of the excavating process. Cavities are often traversed by tough septa and bridles, and are then described as trabeculated. The trabeculæ were formerly said to consist of persistent bronchi and blood-vessels, but they have been shown by Dr. William Ewart to be chiefly composed of condensed airless lung, representing the remains of collapsed alveolar tissue originally separating discrete cavities. The ridges and stumps often observed on the walls of vomicæ are relics of trabeculæ destroyed by ulceration.

In acutely developed and extending cavities the wall is ragged, and formed by soft caseating or necrotic material. Such cavities are commonly filled with thick pus, their walls softened and in a state of purulent infiltration. Chronic and quiescent vomicæ are lined with a definite pyogenetic membrane like that of a chronic abscess. The lung tissue around may be indurated or simply collapsed; less frequently spongy or emphysematous. Extension usually takes place by slow ulceration of individual cavities, which tend ultimately to coalesce; but in some cases acute suppuration and sloughing cause rapid destruction of the lung. Excavation sometimes begins as a tuberculous bronchiolitis, ulceration subsequently extending through the thin bronchiolar wall to the surrounding alveoli. In other cases a dilated bronchus may undergo secondary ulceration and become sacculated. It may be very difficult to decide whether such cavities were originally bronchiectatic or pulmonary. True bronchiectatic cavities are seldom very large, whereas those of pulmonary origin may involve the greater part of a lobe, or even the whole of one lung. Excavations of this magnitude are always the result of fusion of several cavities. In the great majority of cases excavation originates in the lung, and is not bronchiectatic. A fuller treatment of this subject is to be found in the article on "Bronchiectasis" in the present volume [pp. 59 and 74].

In the course of excavation the bronchi become ulcerated and eaten away, so that ultimately their wall passes insensibly into the lining membrane of the vomica. Cicatrisation may cause narrowing or virtual obliteration of the bronchial orifices. Chronic cavities not uncommonly undergo a considerable reduction in size, as the result of contraction of their capsule or of the neighbouring lung. It has even been asserted that they may close completely, but there is no proof of this, and such an event, in view of the imperfect removal of the secretions effected through the bronchi, must be regarded as highly improbable. A vomica resembles a chronic abscess discharging externally through a narrow sinus; now unless the abscess can be freely opened it will not granulate up thoroughly, and will continue to secrete for years. Contraction of a cavity is, however, promoted by a spongy yielding condition of the adjacent lung.

Fibrosis is an essential feature in all chronic excavation, and, as we have seen, the same change is always present in chronic tubercular consolidation. In the most pronounced examples of this condition fibroid induration is associated with excavation. In either case fibrosis causes shrinking of the lung, the upper lobe as a rule being most affected; but at times the whole lung becomes uniformly contracted. In extreme cases the lung may be reduced to the size of a man's fist. Contraction of the lung is followed by elevation of the diaphragm and of the abdominal viscera, displacement of the heart and mediastinum to the affected side, and a varying amount of depression of the chest wall. When the lung is not too firmly adherent to the ribs, a contracting cavity at the apex may shift slightly outwards and backwards towards the fixed point, the root of the lung, as shown by Dr. C. T. Williams.

The primary cavity is situated at the apex of the upper lobe. *Secondary cavities* may be formed in any part of the lung, but Dr. William Ewart pointed out that excavation is especially prone to attack a definite region, the apex of the lower lobe, and at a date anterior to the implication of the lower part of the upper lobe. The base and anterior border of the lower lobe are least prone to excavation, just as these parts are the last to be involved by the disease.

It remains now to consider *the mode in which the tuberculous process extends through the lungs.* In generalised miliary tuberculosis, where infection, for the most part, is derived from a caseous lymphatic gland—that is, from a source external to the lungs—the pulmonary blood-vessels are flooded with bacilli, and the lungs become stuffed with miliary granulations from apex to base. In chronic pulmonary tuberculosis the lungs become gradually but progressively invaded by a process of auto-infection, the primary focus being situated at the apex of one upper lobe. In a moderately advanced case we find one lung more diseased than its fellow, and towards the apex of the former a cavity or cavities with tough walls, the tissue around being pigmented and fibroid, and often containing some caseous nodules. In the lower part of the same lung we see scattered tuberculous nodules and masses, some softening to form small cavities. The other lung presents lesions of a similar appearance and localisation, but in a less advanced stage. It sometimes happens that the disease becomes partially arrested in the lung first attacked, while in the lung secondarily involved it extends progressively from apex to base.

It cannot fail to strike the observer that the secondary lesions in the lung are not the result of direct extension by continuity from the apex, for the individual foci are separated by tracts of healthy lung tissue. Nor is it possible to believe that tuberculosis spreads exclusively or mainly by lymphatic or vascular channels; for, in cases where the disease is not too advanced, the lesions often consist solely of a cavity at the apex of the upper lobe surrounded by a zone of tuberculous infiltration, and some racemose masses of tubercle at the apex of the lower lobe; the rest of the lung being unaffected. Extension to the lower lobe is evidently effected through the bronchial tubes, infective secretion being inhaled from the apical cavity into the bronchi of the lower lobe. This view is in harmony with the results of the "inhalation tuberculosis," artificially produced by exposing animals to a spray of tuberculous sputum. It also accords with the fact that the prevailing lesions in man are broncho-pneumonic in character. Dr. Ewart explains the marked proclivity of the apex of the lower lobe to secondary excavation by the fact that the bronchus supplying this part is a wide, straight tube coming off horizontally from the main bronchus, a condition which appears to favour the inhalation of infective secretion from cavities in the upper lobe. Dr. J. K. Fowler also has pointed out that the distribution of tuberculous disease follows a very definite path. From the initial lesion at the apex the process spreads downwards in the upper lobe. Excavation of this region is followed by secondary disease of the apex of the lower lobe on the same side, and of the apex of the upper lobe of the opposite lung. Dr. Fowler states that the former district is involved before the latter; but in my experience the apex of the opposite lung is quite as often the first point to be affected with secondary disease, though the apex of the lower lobe of the lung primarily attacked is almost always implicated at an early date. The lower part of the upper lobe is then gradually infiltrated, and simultaneously the disease extends from the apex of the lower lobe forward and downward along the interlobar septum. The base and anterior border of the lower lobe are the last parts to be affected.

In the process of destruction *blood-vessels* for the most part become obliterated as the result of thrombosis; but when rapid excavation is taking place, ulceration may extend into large vessels and cause severe hæmorrhage. In cases of a more chronic nature it is not uncommon to find aneurysmal dilatation of branches of the pulmonary artery lying in the walls of a vomica. In my post-mortem examinations aneurysms were found in 15 per cent of all cases of pulmonary tuberculosis; these aneurysms consist of a lateral expansion of the vessel on its exposed side. In rare instances an artery crossing a cavity becomes uniformly dilated to form a fusiform aneurysm. In either case the dilatation is to be attributed to two causes: (i.) to arteritis and softening of the arterial coats, the result of extension of inflammation from the cavity; (ii.) to withdrawal of support from the wall of the exposed vessel. Pulmonary aneurysms vary in size from that of a pin's head to that of a plum, but they are seldom larger than a cherry. It is usual to find only one aneurysm; though, at times, several may be discovered in the same cavity or in different parts of the lung. In one extraordinary case I found twenty-two aneurysms in one lung. Rupture of the sac is a common event, and is by far the most frequent cause of profuse hæmorrhage. In a series of eighty cases of fatal hæmoptysis, examined by myself, a ruptured aneurysm was found in seventy. When rupture does not occur, thrombosis is apt to ensue. Thrombosed aneurysms are often met with in cases where no hæmorrhage has taken place. Observation shows that aneurysms after leaking for some time may become ultimately cured by coagulation of their contents. When the cavity containing the aneurysm is small, the pressure of the effused blood may be sufficient to prevent further hæmorrhage. If the patient live long enough, the healed aneurysm in time undergoes necrosis, and may entirely disappear.

Localised *gangrene* occasionally takes place in connection with rapidly spreading excavation. It is, however, a remarkable fact that, in spite of the existence of numerous profusely secreting cavities, putrid changes are very rarely met with as a result of tubercular disease.

Pleurisy is a well-nigh constant accompaniment of the pulmonary disease, and is mostly due to extension. Pleurisy may also be consecutive to peritonitis, the virus being transmitted from one serous cavity to the other through the lymph spaces of the diaphragm. Primary tuberculosis of the pleura is said to occur, but of this there is some doubt. In cases of apparently primary pleural origin the disease may have started in a small caseous bronchial gland which has escaped detection.

Fibrinous exudation is the commonest form, but sero-fibrinous effusion often ensues. Empyema is uncommon in adults, though less rare in children. Hæmorrhagic exudation is occasionally met with, and may be attributed to rupture of the newly-formed capillaries of the inflamed pleura. In many cases tuberculous granulations and, less frequently, caseous nodules can be recognised in the serous membrane. But it is not infrequently impossible to discover any naked-eye signs of tubercle, whether in cases of fibrinous, sero-fibrinous, or suppurative pleurisy. In some instances of this description the microscope may reveal the presence of isolated miliary tubercles in the thickened pleura. There can be little doubt that the granulations in the pleura, as in the peritoneum, may undergo complete fibrous transformation. It is not improbable that in some instances pleurisy may have a non-tuberculous origin. In any case the ultimate result of pleurisy is to cause more or less thickening and adhesion of the pleura. The former may attain to considerable dimensions in chronic cases, especially at the apex of the lung, where the pleural investment may measure as much as an inch in thickness.

Rapid softening and excavation of the peripheral parts of the lungs are apt to cause perforation of the pleura and entrance of air into the serous cavity, if the pleural space at the affected spot have not previously been obliterated by adhesions.

Pneumothorax causes collapse of the lung, and is followed in most cases by effusion of serous or, more often, of purulent fluid in consequence of the entrance of tubercle bacilli and pyogenetic cocci from the lung.

It is not unusual to discover more than one perforation of the pleura. The opening may be situated at any point where the pleural surfaces are not adherent. The middle third of the lung corresponding to the lower part of the upper lobe and upper part of the lower lobe is the most frequent site of perforation. Occasionally the air escapes into the subcutaneous tissue of the chest wall or into the mediastinum, and surgical emphysema is produced. At times a cavity in the lung may extend outwards through the pleural adhesions and give rise to emphysema, or to an abscess in the chest wall communicating with the lung. Pneumothorax was found in 11 per cent of the cases of phthisis which I examined after death.

The bronchial, mediastinal, and tracheal *glands* are very often the seat of secondary tuberculous deposit. They may also be primarily affected, and, as already mentioned, the disease may extend thence to the lung or pleura. The extreme frequency with which arrested tuberculous lesions, in the shape of calcareous nodules, are found in these glands is well known to all who are in the habit of making necropsies.

Stenosis of a main bronchus is occasionally caused by enlarged glands in children; but this very seldom occurs in adults, as their bronchial tubes are much firmer. The smaller bronchi may be compressed in adults as in children. Marked obstruction entails some degree of collapse of the lung, and sometimes gives rise to bronchial dilatation beyond the seat of pressure. In one case I found that a large calcareous bronchial gland had perforated the bronchus and set up ulceration, which had extended at another point into a larger branch of the pulmonary artery.

Suppurating caseous bronchial glands may perforate the trachea, bronchi, lung, œsophagus, or pericardium. Sudden death has more than once resulted from the entrance of a caseous gland into the trachea. In cases where a fistulous communication is established between the œsophagus and the air-passages a septic broncho-pneumonia ensues, and pulmonary gangrene has been a relatively frequent complication.

From the foregoing sketch it will be seen how manifold are the lesions of phthisis pulmonalis. The unity of phthisis, that is to say, the essentially tuberculous nature of the disease first advocated by Laennec, was long and vehemently disputed; but the truth of this doctrine was at length removed from the sphere of controversy by Koch's discovery of the tubercle bacillus. The presence of the specific microbe in miliary granulations, caseous nodules, caseous pneumonia, and pulmonary cavities supplies a positive demonstration of the pathological identity of these apparently different manifestations. Hence such distinctions as tuberculous, pneumonic, tuberculo-pneumonic, catarrhal and scrofulous phthisis, always artificial and unworkable, are now entirely superfluous. Phthisis is tuberculous disease of the lungs.

SYMPTOMS.—The manner of invasion of pulmonary tuberculosis varies somewhat in different cases. From the slowness of growth manifested by the tubercle bacillus we might expect the invasion of the disease to be gradual. In the great majority of cases this is the case, and certain general or constitutional symptoms often precede those of local disorder of the respiratory organs. But as in a considerable number of cases the disease begins more or less abruptly, we must distinguish (A) acute, and (B) chronic tuberculosis.

A. Acute pulmonary tuberculosis.—Three forms of the acute disease may be recognised.

I. *Lobar-pneumonic form.*—In this form—the rarest of the three—the whole of one lobe, nearly always the upper lobe, or the greater part of one lung becomes converted into a solid gelatinous or caseous substance. The consolidation, though massive, usually presents some scattered foci of older date, suggesting that the diffuse pneumonia is secondary to an originally localised form of tuberculosis.

This sequence of events is well illustrated by cases where a cavity exists in the apex or other part of the lung, under which circumstances the diffused infiltration may be attributed to the inhalation of infective secretions from the cavity. But in a few recorded instances the caseous infiltration has been perfectly uniform, which observations support the belief that the affection was, from the first, lobar and acute, all parts having been simultaneously and equally attacked. In some of these cases there was a cavity in the lung which may have been the starting-point of the pneumonia. Miliary tubercles may sometimes be discovered in the lower lobe or in the opposite lung; caseous nodules are more common. Tuberculous pleurisy, mostly of the dry variety, is a constant accompaniment.

The disease may begin sharply with a rigor, high fever, dyspnœa, pleuritic pain, and a short cough with mucoid, tenacious sputum, which may be rusty or may contain florid blood. Occasionally the attack begins with hæmoptysis. Herpes labialis is not uncommon. The patient often attributes his illness to a chill.

The foregoing mode of invasion closely simulates acute pneumonia. In other cases the onset may be less abrupt, the patient experiencing a malaise, aching in the back and limbs, and slight cough and expectoration, before the onset of marked pyrexia and other pneumonic symptoms. Physical examination discovers signs of consolidation, dulness, tubular breathing, crepitant or subcrepitant râles, bronchophony and increased tactile vocal fremitus. The breath-sounds may be merely weakened, and no tubular breathing may be heard for some time. Pleuritic friction is frequently met with; signs of effusion are somewhat rare. The whole picture is that of acute pneumonia, for which the disease is almost invariably mistaken at first. But no crisis appears, and the fever persists for weeks. In a few instances the temperature becomes lower, and after a few days the symptoms abate somewhat; but the improvement is only short-lived, and the patient relapses into his former condition. The fever for the first two or three weeks manifests a remittent character, the evening temperature being one or two degrees higher than the morning, and ranging from 103° to 104° F. Later the temperature falls somewhat and assumes a hectic character. From the first the patient wastes rapidly and exhibits extreme prostration, sometimes passing into a typhoid state with dry tongue, subsultus, and mild delirium. In the less rapidly fatal cases signs of excavation of the lung gradually come on. The sputum becomes muco-purulent, and is found to contain tubercle bacilli, and perhaps elastic tissue. A fatal termination may be reached in less than a fortnight; more often life is prolonged for six weeks or two months. Now and then the disease gradually loses its acute character and assumes the form of chronic pulmonary tuberculosis.

The diagnosis during the first week or ten days presents great difficulties. In some instances the invasion is less sudden, and the severity of the symptoms less pronounced than in cases of acute lobar pneumonia. But these distinctions are often wanting. In the tuberculous form the fever is less continuous, and is generally marked by irregular remissions.

The pulse-respiration ratio, again, is less deranged than in acute pneumonia; for the pulse-rate is greatly increased, often reaching 130 to 140, with a respiration of 30 or 40.

It has been said that in tuberculous cases the breath-sounds over the affected lobe are more often weak and suppressed than tubular, but this sign is by no means constant; moreover, this sign is not very rare in croupous pneumonia. From acute pneumonia with delayed resolution the disease may be discriminated by the progressive wasting and prostration, as well as by the fluctuating high temperature which accompanies it; for in the former complaint, in spite of the persistent pulmonary consolidation, the general condition mends and the temperature falls. In doubtful cases the appearance of signs of excavation, and, above all, the detection of tubercle bacilli in the sputum, are the only facts on which a positive diagnosis can be based. The complications of this form of tuberculosis do not differ materially from those attending the chronic variety, under which head they will be discussed; but it may be said that complications are much less frequent in acute cases, owing to the rapid termination entailed by the severity of the pulmonary lesions.

II. *Broncho-pneumonic form.*—This form, which is much less uncommon than the last, represents what has been called galloping consumption or phthisis florida. The special anatomical features consist of disseminated tuberculous foci, of various sizes, which may be soft, yellowish white, and cheesy; or grayish, slightly pigmented, of racemose shape, and somewhat indurated. Miliary tubercles are seldom to be seen. In most cases rapid softening and excavation of the nodules is a very prominent feature. Small suppurating cavities with soft ragged walls are scattered through both lungs. In the apices of the upper lobes the cavities are generally larger, and in some cases the apex is the seat of old fibrosis and excavation. The lung tissue separating the nodules is often hyperinflated, especially towards the anterior borders; in other parts the nodules are embedded in tracts of grayish red consolidation, more particularly towards the back. This fusion of the individual foci may ultimately result in a diffuse infiltration of lobar dimensions. The bronchi are always deeply injected and contain abundant purulent secretion. The localisation of the lesions is essentially broncho-pneumonic and lobular, and depends on the inhalation of tubercle bacilli from a cavity in the lung or from external sources. Pleurisy in some form, whether dry, sero-fibrinous, or sanguineous, is always present. The larynx and large air-passages are more prone to tuberculous ulceration than in the lobar-pneumonic form, in consequence of the more profuse secretion discharged from the cavities and bronchi in the present variety.

The mode of onset is subject to considerable variations. Occasionally without any early period of ill-health the patient is suddenly seized with rigors and other symptoms of acute pneumonia: more often the disease begins insidiously with a cough, which, after the lapse of a few weeks, is succeeded by fever, malaise, and other constitutional symptoms. Hæmoptysis is occasionally the first symptom. In some instances the disease begins with symptoms of gastric disturbance, loss of appetite, furred tongue, and vomiting; and the real nature of the malady is not recognised until the chest is examined.

In recent years this form of tuberculosis has not uncommonly followed an attack of influenza. Whatever the mode of invasion, marked wasting and loss of strength soon appear. Hæmoptysis is not very common, and is seldom profuse. The sputum at first is muco-purulent, but it soon becomes more puriform, and sometimes acquires a greenish yellow colour; in some cases it has a reddish brick-dust colour for weeks. Tubercle bacilli and elastic tissue are generally recognised before long. Night sweats are frequent, and often are very profuse. The temperature ranges high, reaching 104° F. at times; the fever is fluctuating, being marked by morning remissions of one or two degrees: as the disease proceeds, the temperature becomes more hectic. Anorexia, vomiting, aphthous stomatitis, a dry red tongue, and diarrhœa are very common, and the patient is apt to pass into a typhoid state. In the worst cases death ensues in three or four weeks, but the end is more often deferred for three or four months. Very occasionally the acute progress of the disease is stayed, and the patient lingers on for eight or nine months.

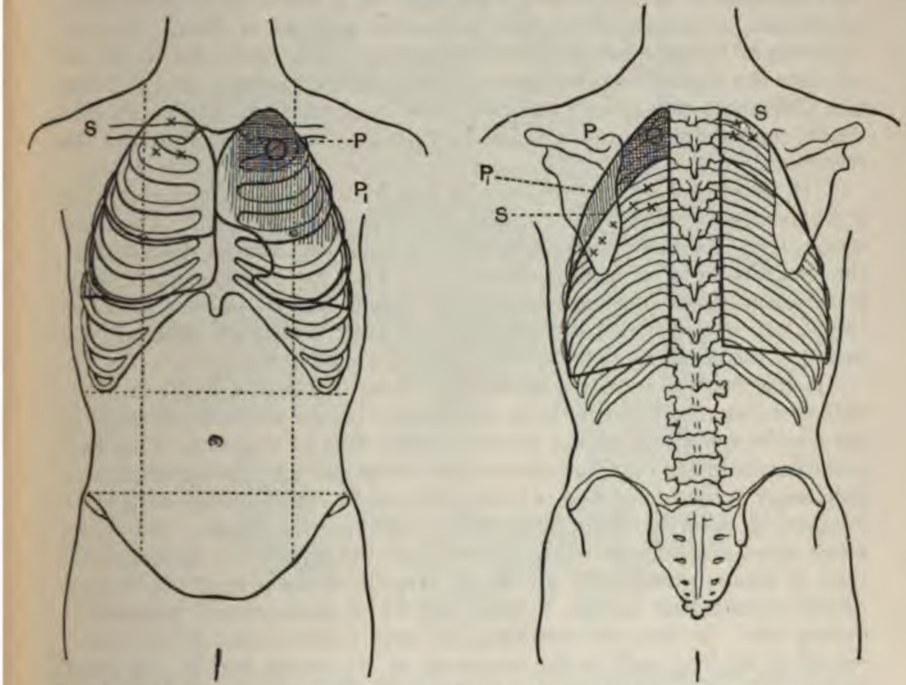

FIG. 7.—Diagram illustrating the localisation of tuberculous lesions in the lungs. P, Primary lesion (double shading); P_1, local extension from primary lesion (single shading); S, secondary lesions.

Physical examination at first reveals nothing more than signs of general bronchitis; but subsequently pleuritic friction and patches of dulness on percussion, more particularly at the apices, make their appearance, and signs of excavation may ultimately be discovered. In some instances the signs may predominate at the base of the lower lobe. In the most acute cases no cavernous signs can, as a rule, be recognised, as death takes place before the cavities have reached sufficient size to permit of their detection. Moreover, the patches of distended lung tissue which separate individual foci tend to obscure the existence of extensive disease. The diagnosis at first rests on the discovery of physical signs of broncho-pneumonia, accompanied by great prostration and loss of flesh. But the detection of tubercle bacilli in the sputum may alone enable us to decide whether the disease be tuberculous or not. In the case of young children the differences are greatly enhanced, for no sputum is obtainable, and death commonly takes place before softening and excavation can be recognised.

III. *Acute miliary tuberculosis.*—In this form the pulmonary condition is frequently dwarfed by the symptoms of general infection. This condition, from its resemblance to typhoid fever, is sometimes described as the typhoid form of acute tuberculosis. In other instances the disease manifests a special incidence on certain organs, and types have been distinguished varying with the parts of the body principally affected; for instance, the cerebral, the abdominal, and the pulmonary.

It has been the custom to draw a sharp distinction between acute miliary tuberculosis and phthisis on account of the marked difference in the clinical symptoms of the two affections; but an eruption of miliary granulations in the other organs is a fairly common complication of chronic pulmonary tuberculosis, and is to be attributed to the entrance of a large number of tubercle bacilli into the pulmonary circulation. Moreover, many cases, clinically indistinguishable from the typhoid or disseminated type of acute tuberculosis, are found after death to present old circumscribed tuberculous lesions of the lung, which had escaped recognition during life. In fact, the acute miliary form differs from chronic tuberculosis of the lung only in the acuteness of its course and in the more widespread infection of the body. In the pulmonary type, which alone will be considered here, the disease may advance in an acute or subacute manner without any premonitory symptoms. In a large proportion of cases a period of ill-health, of variable duration, precedes the onset of the disease. The symptoms first noticed are cough, expectoration, dyspnœa, and occasionally pleuritic pain. Dyspnœa, as a rule, soon becomes the predominant feature, and is often accompanied by marked cyanosis. Hæmoptysis is uncommon; but now and then it is the earliest symptom. The temperature is generally high, reaching 103° to 104° F., and the morning remissions are less pronounced than in the broncho-pneumonic form.

Some cases have been known to run their course without any definite elevation of temperature. The pulse, from the first, becomes rapid and weak. Examination of the chest reveals signs of general bronchitis, fine bubbling râles, and rhonchi on both sides. At first no dulness on percussion can be elicited, but the anterior parts of the lungs are found to be rather hyper-resonant, the change depending on compensatory distension of the alveoli—the so-called "acute emphysema." As the disease progresses, pleuritic friction sounds are often heard; and patches of dulness pointing to secondary broncho-pneumonia may sometimes be recognised. In these parts the breath-sounds may be tubular, but more often become muffled. This difference does not depend on the preponderance of consolidation or pleural effusion; for, in the absence of pleuritic exudation, the vesicular breathing may be greatly diminished by the concomitant bronchitis and lobular collapse. Lobar pneumonia is an occasional complication. The patient rapidly loses flesh and strength, dyspnœa and cyanosis increase, the cough grows more troublesome, and the sputum—which at first was mucoid—now becomes muco-purulent. It is rare to find tubercle bacilli in the expectoration, and when this happens, a cavity, often a very small one, will generally be found in some part of the lung. The spleen is more or less enlarged, and may sometimes be recognised by palpation.

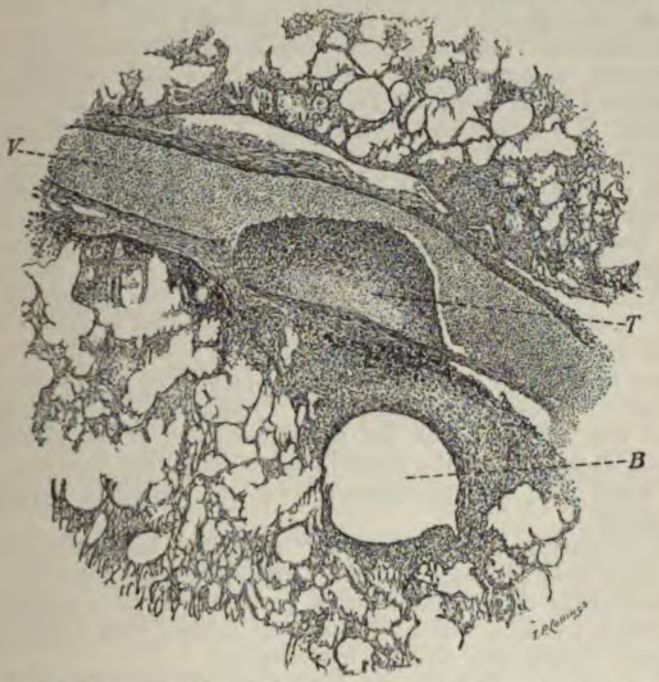

FIG. 8.—Photograph of a section of the lung from a case of acute miliary tuberculosis, showing the mode in which general infection occurs through branches of the pulmonary veins. (Low power.) V, Small pulmonary vein; T, tubercular growth from intima, projecting into the lumen of the vein; B, small bronchus showing tubercular infiltration of its walls.

The diagnosis is occasionally easy, but more often difficult. In the presence of general bronchitis, associated with marked dyspnœa, cyanosis, pyrexia, and rapid emaciation, the diagnosis presents little difficulty. But in cases where the evidence of bronchitis is slight or absent, the disproportionate amount of dyspnœa is a diagnostic point of considerable value. Tubercles may occasionally be recognised in the choroid by means of the ophthalmoscope, and tubercle bacilli have, in a few instances, been found in the blood; but unfortunately such evidence is rarely to be obtained.

B. Chronic pulmonary tuberculosis.—The following modes of invasion may be recognised in their order of frequency:—

(i.) *Insidious.*—The commonest prodromal symptoms are loss of flesh and strength, accompanied, in some cases, by a slight evening rise of temperature. Less frequently the disease is ushered in under the guise of anæmia, or of a functional derangement of the digestive system.

(ii.) *Bronchitic.*—After frequent attacks of bronchial catarrh, or without any previous tendency to bronchitis, the disease begins with cough and expectoration, which are attributed at first to a common catarrh; but after a few weeks or months pyrexia and other constitutional symptoms make their appearance. In some instances careful inquiry will establish the fact that a period of ill-health existed before the appearance of the cough. Many cases with such a story have originated in influenza.

(iii.) *Pleuritic.*—The first definite symptom is pain of pleuritic type, increased by cough or deep inspiration. The pleurisy is generally of the dry form, but effusion may take place. Pyrexia and other symptoms of phthisis may follow hard on the pleuritic seizure, or the pleurisy may gradually disappear, and the patient make a temporary recovery, only to fall ill again later, with pronounced symptoms and signs of pulmonary tuberculosis.

(iv.) *Hæmoptoic.*—In this class, the "phthisis ab hæmoptoe" of the old authors, the first symptom to attract attention is hæmoptysis. When the hæmorrhage is profuse, it almost certainly indicates rupture of an aneurysm in a vomica, that is, old-standing disease; even when no other evidence of a pulmonary lesion is forthcoming.

(v.) *Laryngeal.*—Phthisis occasionally begins with laryngeal symptoms; hoarseness, loss of voice, hyperæsthesia, or paræsthesia of the throat being the most common.

Symptoms.—A constant and perhaps the most important symptom from the diagnostic point of view is *cough.* At first dry, short, and infrequent, it is accompanied, sooner or later, by expectoration, and may become so incessant as to prevent sleep and to set up vomiting, whereby the patient's strength becomes reduced in the most serious manner. There is no direct relation, however, between the gravity of the disease and the severity of the cough. Some patients, with extensive pulmonary lesions, have little or no cough; whilst in others, with comparatively slight disease, cough may be the predominant symptom. Cough is generally most troublesome in cases of progressive disease, and where the larynx, trachea, and large bronchia are actively engaged; but it depends to a considerable extent on the excitability of the nervous centres. When the larynx is extensively affected the cough is peculiarly muffled and hoarse. In some cases, especially when large cavities form in the base of the lung, it assumes a paroxysmal character. Coughing fits occur most frequently in the early morning, owing to the accumulation of secretion in the larger air-passages during the night. In some cases an irritable cough is excited by the ingestion of food, and the fit may end in vomiting. This occurrence is partly to be explained by the mechanical compression of the stomach and abdominal viscera against the diaphragm; but vomiting so often follows slight fits of coughing that it seems necessary to assume the existence of a neurosis of the vagus in these patients.

Expectoration.—In the early stages expectoration is scanty and mucoid; but it soon becomes muco-purulent, and is commonly very viscid. At times it is thin and watery, from admixture with saliva. As the disease progresses, the sputum collects into small thick lumps of a dirty white or yellowish colour; this "nummular sputum" is more common where cavities have formed in the lung, but it may be met with in cases of simple bronchitis and of bronchiectasis. It is not uncommon in the same specimen to find small yellowish spots or streaks mixed with frothy mucous secretion —the mixture representing bronchial secretion and pus from cavities in the lung. At times, especially in advanced cases, the sputum becomes uniformly opaque and thick, and may assume a greenish colour. Expectoration is sometimes markedly paroxysmal, especially where cavities exist in the lower part of the lungs. Blood is often discharged with the sputum. The blood-stained sputa may be bright red, or, when blood-clots have been retained in cavities or bronchi for some time, the colour may be dark purple or blackish. In certain instances the sputum presents a brownish or chocolate colour from decomposition of blood in the cavities or bronchi. In other instances it may be of a brick-red tint, especially when active ulceration of the lung is going on; but it seldom has the rusty, tenacious character of acute pneumonia. Fœtor is practically unknown in the absence of such complications as bronchiectasis or gangrene. In the more chronic forms of phthisis small particles of calcareous matter, consisting mainly of phosphate of calcium, are coughed up from time to time. These pulmonary calculi represent caseous material that has undergone calcification, and has become loosened in the process of excavation. Sometimes they show a tendency to branch, so that some think that they may come from the small bronchia. I have found them on many occasions in the recesses of old cavities in the lung. Calcareous bronchial glands occasionally perforate the air-passages and are expectorated. The discharge of pulmonary calculi implies ulceration of the lung or air-passages, and is a sign of chronic disease; but no further diagnostic value can be assigned to it.

As regards the importance of the sputum of phthisis, it must be allowed that the naked-eye appearances alone possess no certain and pathognomonic significance, if we except the presence of blood and calcareous matter. The former will be considered under the head of hæmoptysis. It is doubtful whether chalky masses are expectorated in any disease other than tuberculosis of the lungs or bronchial glands; consequently this event has a certain diagnostic significance. Microscopic examination of the sputum is a most valuable method of diagnosis. By this means we recognise various forms of cells—squamous, flattened, spheroidal, columnar and ciliated epithelium, blood corpuscles, pus cells, mucin, crystalloid products of chemical change, such as cholesterine, leucine and tyrosine, fatty acids and drops of myeline, carbon particles, elastic tissue from the lungs, and microbes of different kinds. Of all these constituents of the sputum two only are pathognomonic, elastic tissue and tubercle bacilli. The presence of the former is a positive proof of destructive disease of some portion of the respiratory apparatus, though it does not enable us to distinguish the precise nature of the disease; but as tuberculosis is by far the commonest ulcerative affection of the lung, the presumption is in favour of this being the process at work. When the elastic tissue shows an alveolar arrangement we may be certain that it is derived from the lung; but isolated fibres may possibly come from the larynx, or from the trachea and bronchi; though, unquestionably, their main source is the lung.

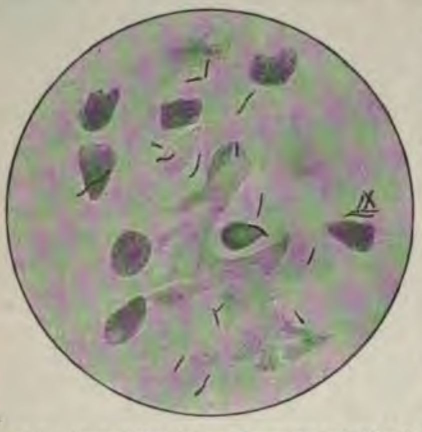

FIG. 9.—Sputum showing pus corpuscles and tubercle bacilli; some of the bacilli are beaded. ×550.

Elastic tissue.—If the opaque whitish particles seen in the sputum be teased out with needles and examined in a drop of water, branching elastic fibres with their curled-up ends, or portions of the more characteristic alveolar framework of the lung, may be recognised under a low power. Sometimes, in cases of chronic excavation, the fibres are encrusted with minute particles of lime salts. The persistent presence of elastic tissue with alveolar grouping is a sign of progressive destruction of the lung. A better and more certain method is that devised by Dr. Fenwick. The sputum is mixed with an equal quantity of a solution of caustic soda of the strength of 20 grains to the ounce, and boiled for a few minutes until the mixture becomes clear. The fluid is now allowed to stand in a conical glass for a few hours, when the elastic fibres fall to the bottom. A drop of the sediment is then withdrawn with a pipette and examined for elastic tissue under a low power (90 to 100). It is important not to continue the boiling too long, as the elastic fibres themselves ultimately become much altered.

Microbes—Tubercle bacillus.—The sputum voided in the early morning

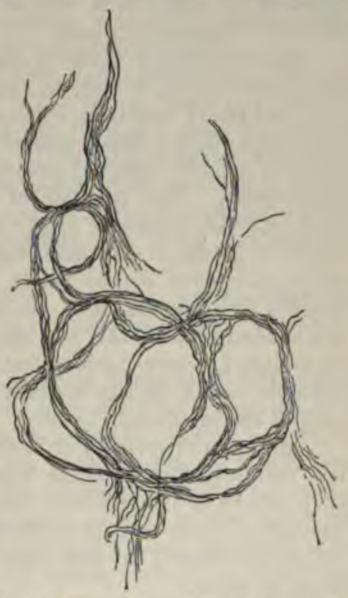

FIG. 10.—Elastic tissue from the lung, with well-marked aveolar arrangement. Perpared from sputum by Fenwick's method. (Low power.)

should, if possible, be chosen for investigation, as it contains no particles of food, and as, being composed of the secretions accumulated during the hours of sleep, it represents a mixture of the products of the various sections of the respiratory tract. The expectoration is poured out into a flat glass dish, and examined against a dark background. The small opaque specks and streaks, or, where the sputum is uniformly opaque, the most curdy portions, are the most suitable for examination. A small portion should be removed with a scalpel, needle, or platinum wire and transferred to a perfectly clean cover-glass. A second cover-glass is pressed gently on the first, so as to distribute the sputum in as thin a layer as possible; and the two glasses are then separated by a sliding movement and allowed to dry. When quite dry, the cover-slips are seized with a forceps and passed three times quickly through the flame of a Bunsen burner or of a spirit-lamp to coagulate the albumin. Various methods are in use for staining the bacillus. Ziehl's modification of Ehrlich's method gives excellent results and will alone be described. The following reagents are required:—(*a*) Ziehl's solution of carbol-fuchsine, consisting of 10 c.c. of a saturated alcoholic solution of fuchsine, added to 90 c.c. of a 5 per cent watery solution of carbolic acid. (*b*) A 25 per cent solution of sulphuric acid. (*c*) A concentrated aqueous solution of methylene blue. The staining fluids should be filtered before they are used. The cover-glasses are placed in some of the fuchsine solution (*a*) in a watch-glass or porcelain dish, which is then heated carefully over a spirit-lamp or a sand-bath until bubbles are given off. The staining fluid is then set aside for two or three minutes to cool. Next, the cover-glasses are removed with forceps and passed through some of the acid solution (*b*) for a few seconds, until the red colour changes to a yellowish gray. The preparations are then washed in a gentle stream of water running from a tap for eight or ten seconds, when the sputum again turns red. Lastly, the slips are stained with a drop of the blue dye (*c*) for half a minute to a minute, washed again in a stream of water for a few seconds, and the excess of water allowed to drain away. The cover-glasses may now be left to dry; or rapid drying may be effected by pressing them gently between two pieces of clean blotting-paper. When they are quite dry the preparations are mounted in a drop of Canada balsam, dissolved in xylol or benzol. If the illumination be good, the tubercle bacilli can be recognised with a magnifying power of 300; but in order to obtain satisfactory results it is well to use Abbé's substage condenser and a one-twelfth oil immersion lens.

With the above mode of staining the tubercle bacilli appear as delicate rods of a red colour, in length from a quarter to a half the diameter of a red blood corpuscle, often straight or slightly curved, and in many cases presenting a finely-beaded appearance. This beading, supposed by some to depend upon the presence of spores, possesses no special clinical significance. The number of bacilli in the sputum is very variable. They may be scattered singly through the preparation, or they may be found in groups. In some instances the sputum seems to be an almost pure cultivation of the bacilli. Many other microbes, pyogenetic and putrefactive, the nuclei of pus and epithelial cells and threads of mucin are stained blue by this process, but no definite importance can at present be assigned to any microbe but the tubercle bacillus. The detection of tubercle bacilli in the sputum is a certain sign of tuberculosis of some part of the respiratory tract.

Instances of primary tuberculous ulceration of the larynx or pharynx are infinitely rare, but the number of bacilli shed from the surface of such ulcers is insignificant when compared with the enormous masses discharged from cavities in the lung. When the sputum contains a large number of bacilli we may reckon on the existence of a vomica, whether large or small.

Hæmoptysis is one of the most striking symptoms of the disease. Streaks of blood may be seen in the expectoration of many other affections, and are the result of capillary hæmorrhage from the lungs or air-passages: such streaks are seldom of much importance; nevertheless, they occasionally herald the approach of a more profuse hæmorrhage. When the amount of blood brought up is considerable, the significance is far greater.

Hæmorrhage from the lungs may occur as the result of hyperæmia and rupture of capillaries, or of gross pulmonary lesions involving perforation of vessels of considerable size. Slight attacks of hæmoptysis are mainly due to capillary hæmorrhage from the lungs, less frequently from the large air-passages, and are indicative of inflammatory or congestive states. When, however, the blood expectorated can be measured by ounces, the bleeding must be attributed to rupture of an artery or vein of some size. Perforation of vessels, generally of an artery, may be effected in three ways:—(α) The walls of the small pulmonary arteries and veins may become infiltrated with a tuberculous growth. The usual consequence of this change is thrombosis of the affected vessel; but in the case of the arteries, softening of the vascular wall may lead to rupture, and some of the small hæmorrhages of phthisis are probably thus produced. (β) The ulcerative process associated with excavation of the lung may perforate an artery of considerable size, and occasion alarming hæmorrhage. It seems strange, at first sight, that this does not happen more often; but the tendency in all but the more rapid forms of tuberculous destruction is towards thrombosis of vessels. This is a more important cause of hæmoptysis than the preceding, but it is very much less common than the next (γ), namely, rupture of an aneurysm in a cavity in the lung: this is by far the most common cause of profuse hæmorrhage.

Hæmoptysis may prove directly fatal from cerebral anæmia, though a termination by syncope is uncommon. The usual cause of death is asphyxia, which results from flooding of the bronchia with the effused blood. Ruptured aneurysms may become closed by thrombosis and the patient recover. There is every reason to believe that most cases of profuse hæmoptysis which end in recovery are due to the rupture of an aneurysm, and that ulceration of large vessels is a much less frequent cause.

The old view that the extravasation of blood can set up inflammatory and destructive changes in the lung—"phthisis ab hæmoptoe"—is no longer entertained.

One of the points adduced by Niemeyer in support of this notion, namely, that pyrexia often appears a few days after the hæmorrhage, is more easily explained by the aspiration of infective cavity secretions, mixed with blood, into other parts of the lung, leading to tuberculous broncho-pneumonia. An attack of hæmoptysis is occasionally determined by some obvious cause of vascular excitement, such as mental agitation, muscular exertion, straining at stool, or menstruation; but more often the patient suddenly begins to cough up blood without any warning, often while in bed. Hæmoptysis is generally repeated frequently, and may last for hours or days with intermissions. The blood expectorated is generally bright and frothy; but when it has gathered slowly in cavities or in the bronchial tubes it may be dark and clotted. The quantity lost varies considerably; as much as two or three pints may be brought up in a short time. When the flow is not excessive the blood is often mixed with sputum, a point of considerable diagnostic importance; and in most cases expectoration of blood-stained secretion continues for a day or two after all active hæmorrhage has ceased. The bleeding may manifest a marked tendency to recur at intervals for some time; in such cases the rent in the walls of the aneurysm has undergone only partial repair, and leaking goes on from time to time. The patient is almost always greatly alarmed by the supervention of hæmorrhage. The face is pale and bedewed with sweat, the extremities cold, and the pulse is feeble; the bodily temperature sinks below the normal. Blood is brought up with a frequent short cough, and is often swallowed. When the hæmorrhage is arrested the temperature returns to the normal range, and on the third or fourth day may rise three or four degrees. After the attack, patients are much exhausted and depressed; partly in consequence of the loss of blood, but still more as the result of nervous shock.

Some patients show no serious deterioration of health after the immediate debilitating effects of the hæmorrhage have passed away; but in not a few instances, under the influence of repeated attacks of hæmoptysis, chronic disease assumes a subacute, progressive character, a result attributable to the violent inspiratory efforts provoked by the presence of blood in the bronchia and the consequent insufflation of infective secretion into healthy lung.

Some writers have described a special variety of phthisis under the name "hæmorrhagic," but there does not appear to be any sufficient reason for the subdivision. Cases beginning with a sudden hæmoptysis, repeated, perhaps, at intervals for a considerable time, may subsequently run the ordinary course of chronic phthisis without any further hæmorrhage. Other patients presenting the usual form of disease may succumb after a succession of attacks; or their first hæmorrhage may prove fatal. Hæmorrhage is an accident which may complicate any case of the disease, and is not a satisfactory basis for classification.

Dyspnœa.—A subjective sense of dyspnœa is seldom complained of, save in the later stages of the disease; though most patients with progressive phthisis exhibit increased frequency of respiration, especially on slight exertion. The rate of respiration rises slightly in the evening. The absence of dyspnœa is explained by the tolerance acquired during the slow, insidious progress of the pulmonary affection, and also, as has been suggested, by the low standard of respiratory requirement due to the reduced volume of blood.

When chronic lesions are complicated by acute tuberculosis, especially in its miliary form, and when pneumothorax occurs, urgent dyspnœa may arise.

Pain in the chest.—Many patients have pain in the chest, mostly in the axillary or mammary regions, varying in degree from a slight aching sensation to the agonising stitch of pleurisy. Severe pain is nearly always referable to the implication of the pleura, in which case tenderness to percussion is often met with. Vague rheumatoid pains in the chest have been regarded as very significant; but in the absence of other symptoms more directly pointing to the lungs, little importance can be attached to them. They are, not infrequently, of muscular origin, and may be attributed to the violence of the cough. Dragging pain over chronic cavities, associated with retraction of the chest wall, is sometimes a persistent symptom depending on stretching of the adjacent pleura and intercostal nerves. The muscles of the chest wall in some cachectic patients are extremely tender to percussion, and the slightest tap may promote muscular contraction; but this increased excitability of the muscles is not peculiar to the disease.

General symptoms.—Pyrexia is a symptom hardly less significant, from the point of view of diagnosis, than cough; and of infinitely more value as a measure of the activity of the disease. The cause of the elevation of temperature must be ascribed to the presence in the blood of some soluble poison produced by the bacillus. It is generally agreed that the pyrexia of tuberculosis attains its maximum, and may often be exclusively present, in the post-meridian hours of the day. A slight evening rise of temperature may be one of the earliest symptoms. Some observers have noted a persistently subnormal temperature as the first definite indication of the disease. Accordingly careful thermometric observations, night and morning, should be made in all cases of obscure ill-health, especially in young persons.

The onset of fever is sometimes accompanied by slight shivering, but a marked rigor is seldom observed except in acute cases. The maximum temperature is found from 2 to 10 P.M., and the minimum from 2 to 8 A.M. In exceptional cases the order is reversed, the morning temperature being higher than the evening—the "inverse type." This may be only temporary, or the same relation may be preserved throughout the whole course of the case.

Two main forms of pyrexia may be distinguished, the intermittent and the remittent. One or other form may predominate or prevail exclusively for weeks or months, but various combinations are apt to arise; in fact, one of the characteristics of tuberculous fever is its fluctuating and irregular course.

In the first or intermittent type the temperature is normal or slightly subnormal in the morning, and reaches 100° to 103° F. in the evening. In the higher grades of this form the fall is still more pronounced, and may amount to 7° or 8° F., the thermometer sometimes registering a temperature as low as 94° or 95° F. The second or remittent type shows a maximum temperature of 103° to 104° F., the minimum temperature being 2° to 3° lower, but not reaching the normal level. A slight degree of intermittent fever, where the maximum, for the most part, does not exceed 101° to 102° F., is often found in the early phases of the complaint; but a similar temperature curve may be recorded at any stage. When the range of temperature is greater, and more particularly when the morning reading is below normal, profuse sweating is very common, and the resemblance to the hectic fever of pyæmia is very close. The remittent form of fever commonly betokens active tuberculous infiltration, and is more often met with in the acute varieties of tuberculosis, but may also appear temporarily in chronic phthisis as the result of acute exacerbations or of intercurrent disease. In acute miliary tuberculosis, uncomplicated by suppuration in the lung or elsewhere, the type of fever is generally remittent, a fact which would point to this being the form of pyrexia peculiar to tuberculosis.

Pulmonary phthisis never runs its whole course without fever, but in many chronic cases there may be no appreciable rise of temperature for long periods of time. Observations by Dr. C. T. Williams have shown that pyrexia may be absent even when the disease is making rapid progress. But this is a very unusual course, and it may be stated as a general principle that activity of the disease is indicated more surely by pyrexia than by any other symptom or sign.

A high evening temperature with a markedly subnormal morning temperature (95° to 97°) is a common feature of advanced and progressive disease, though in such cases the fever may assume the remittent type at any time. Towards the close of life the temperature generally tends to fall. The very low temperature registered in pneumothorax, in some cases of excessive pulmonary hæmorrhage, and in the comparatively rare instances where perforation of the intestine occurs, must be attributed to the effects of shock. When we consider the various processes of infiltration, necrosis and suppuration occurring in the lungs, as well as the numerous complications that may arise, we cannot be surprised at the great variations exhibited by the temperature chart.

It is necessary to mention the assertion of Peter, that the temperature of the skin differs on the two sides of the thorax, the higher reading being found on the side corresponding to the lung more affected. Most observers have failed to verify this statement, and a similar want of symmetry in the temperature of the two armpits has occasionally been observed in other conditions.

Sweats.—Profuse perspiration is a common symptom in pyrexial cases, though it has no constant relation to the fever. Sweating is most pronounced in the early hours of the morning, when the temperature of the body is at its lowest; but it also occurs sometimes while the fever is continuously high. Night sweats may occasionally occur in apyrexial periods, in which case they seem to be due to fits of coughing.

Dr. Lauder Brunton has suggested that sweating is the result of exhaustion of the respiratory centre in the medulla, and consequent accumulation of carbonic acid in the blood; the effect of this being to stimulate the sweat centres. This symptom is certainly more prevalent in advanced cases, associated with excavation and suppuration of the lung; but it is not uncommon in early and circumscribed disease, in which case Brunton's hypothesis seems less applicable.

Emaciation is one of the most frequent and important symptoms, and may proceed to an extreme scarcely reached in any other disease; hence the names phthisis and consumption.

The greatest loss of weight is witnessed in chronic cases, but although sometimes at first comparatively slight in the acute type which terminates in a few months, it is never absent altogether. Wasting affects all the soft parts, but especially the fatty and muscular tissues. It has been said that the liver does not share in the general wasting, but this statement is probably to be explained by the great liability of the liver to congestion and to fatty and amyloid degenerations, conditions which involve enlargement of the organ. The loss of flesh is mainly, though not exclusively, due to the increased metabolism inseparable from the febrile process. Patients with a high temperature lose weight as long as the fever continues; and, as a rule, when the heat of the body becomes normal, wasting ceases. Moreover, a certain correspondence between the degree of the fever and the loss of weight can often be recognised. At the same time a considerable degree of fever is not incompatible with an actual increase in weight, if an adequate supply of food can be taken, and if digestion and absorption be unimpaired. In apyrexial cases the weight of the body may remain stationary for months, or even years; but when pyrexia appears, loss of flesh soon follows.

The early emaciation, which not uncommonly precedes any appreciable rise of temperature or other signs of disease, cannot be thus explained. In the absence of any definite knowledge on this point we may adopt the provisional hypothesis that the toxins of tuberculosis may cause a general failure of nutrition apart from any febrile movement. Functional derangements of the stomach and diarrhœa, by their interference with digestion and assimilation, are potent causes of wasting.

Debility.—A sense of weakness and loss of energy, both of mind and body, are commonly felt at a very early date, and not infrequently appear to be out of all proportion to the extent of the disease.

Anæmia.—In certain patients the complexion acquires a peculiar faded yellowish tint, which has been well likened to a dead leaf. On examination the blood shows the changes of chlorosis—a considerable reduction of the hæmoglobin with a relatively slight diminution in the number of the red corpuscles, and also a diminution in mass. In active pyrexial disease a moderate degree of leucocytosis is common.

The pulse in all progressive cases is rapid and of low pressure; sometimes it is full, but more often small. The frequency of the heart's action is not invariably determined by the degree of fever, but seems rather to stand in direct relation to the extent and activity of the disease, and to the strength of the patient; consequently the pulse is a most valuable index of the gravity of the case. The pulse is generally somewhat more frequent in the evening, but exceptions to this rule are met with. Some authors have regarded a persistent rapidity or ready excitement of the heart as important premonitory symptoms; and there is no doubt that cardiac erethism is often present at a very early stage of the disease.

Cyanosis is seldom a marked symptom, except as the result of serious pulmonary or cardiac complications, though the fingers, toes, lips, ears and nose often present a slightly dusky or livid hue, in marked contrast to the general pallor. Coldness of the extremities and extreme sensitiveness to trifling depressions of temperature are a common complaint, and further testify to the feebleness of the circulation.

Skin and hair.—In connection with the subject of nutrition reference must be made to the state of the skin and hair. The skin of tuberculous patients is generally very oily, and the sweat has a peculiarly pungent garlicky odour. In some emaciated subjects, on the contrary, a dry branny condition, "pityriasis tabescentium," may be observed. The texture of the skin in one type of patients is delicate and thin, and the complexion transparent; while in another class the skin is coarse and the complexion dull and muddy,—distinctions which are included in Sir William Jenner's classical description of the tuberculous and scrofulous diatheses respectively. But in the majority of phthisical persons no such peculiarity can be recognised, though in all cases of long-standing some degree of pallor is wont to appear. Pigmentation of the skin may become so marked in certain chronic cases that Addison's disease may be simulated; but the patchy pigmentation of the tongue and buccal mucous membrane, so characteristic of the latter affection, does not occur. The cause of this pigmentation is unknown. "Pityriasis versicolor" is observed rather frequently on the chest and back, but no special significance can be assigned to this parasitic complaint. Lupus is only occasionally found in phthisical patients. The terminal phalanges of the fingers and toes frequently show a curious bulbous enlargement associated with incurvation of the nails; the swelling is believed to be due to thickening of the subcutaneous tissue, but it is possible that the bony structures may also be involved. This clubbing of the fingers and toes is not peculiar to tuberculosis, and it is found in empyema, in chronic pneumonia, in certain forms of heart disease, and in emphysema—conditions in which impediment of the pulmonary circulation and consequent engorgement of the systemic veins are a common factor.

The hair, participating in the general malnutrition, may become thin and straight; but this change is by no means constant, as in some persons the hair of the head and beard remains very thick, and the trunk may be unusually hirsute. In children and young persons the body is sometimes covered with a growth of fine downy hair.

Physical diagnosis.—Certain abnormal forms of chest are met with in many phthisical subjects. Two special varieties may be mentioned on account of the frequency with which they occur. In the first, named alar or pterygoid by Galen and Aretaeus and in our own day by Dr. Gee, the angles of the scapulæ project like wings, the ribs are unduly oblique, the shoulders fall, and the length of the thorax from above downwards is increased, but the antero-posterior diameter is small. In the second or flat type the chest in front is flat instead of being rounded, and the sternum may even be depressed below the level of the costal cartilages, which lose their curve and become straight. These peculiarities are certainly common in tuberculous persons, but they are frequently met with also in persons who remain free from the disease. Moreover many phthisical persons have large and well-formed chests. It cannot be said, in other words, that there is any type of thorax peculiar to phthisis, although the chest, in common with the muscles and bones, is often ill developed. Much more importance is to be attributed to partial deformities of the chest walls, the result of pulmonary disease.

Before discussing the physical diagnosis of the disease in its early stages, it may be well to recall briefly a few anatomical facts. The initial lesion consists of a small nodule or group of nodules situated somewhat below the extreme apex of the lung. The nodule is bronchopneumonic; that is, it consists of a localised bronchitis with surrounding lobular consolidation. The neighbouring parts of the lung at first remain spongy and practically unaltered, so that the nodule is enclosed in a shell of healthy pulmonary tissue.

Physical examination of the lungs at this period may yield a completely negative result, especially when the focus of disease is small, and the layer of spongy lung around is fairly thick. As long as the surrounding lung is crepitant, percussion gives no dulness. The earliest signs are almost exclusively discovered by auscultation, though at times, on inspection and palpation, a slight diminution of respiratory movement may be recognised in the subclavian region. Owing to the persistency of the apical catarrh, and to the consequent lobular collapse, the entry of air into the corresponding section of lung is diminished, and the breath-sounds become weakened. Jerky, interrupted, or wavy breathing—the "respiration saccadée" of the French—is not very uncommon, but is not pathognomonic, and may often be heard in neurotic or hysterical, or even in healthy persons. Weakness of breathing at the affected apex is often associated with increased loudness of the vesicular murmur on the opposite side—a condition known as compensatory or puerile breathing, which is sometimes erroneously regarded as an indication of disease.

Another important and early sign is furnished by harshness of the breath-sounds affecting the expiratory sound to a greater degree, and at an earlier date than the inspiratory. The expiratory murmur at the same time acquires a higher pitch, and becomes so prolonged as to equal or exceed the length of the inspiratory sound. This change is an early indication of consolidation, the character of the breath-sounds being modified, without having actually attained to the bronchial or tubular type. It is necessary to distinguish this condition from mere prolongation of the normal expiratory murmur, which may be the result of bronchial obstruction, as in bronchitis, emphysema and asthma, and is then generally associated with a weak vesicular inspiration. At this period the vocal resonance and tactile fremitus may be slightly increased, or there may be no recognisable alteration.

It will be convenient at this point to make a passing reference to what may be called the physiological dissimilarity of the right and left apices. In the large majority of healthy persons, especially in thin subjects, the breath-sounds are louder, the expiratory murmur more audible and prolonged, and the vocal resonance and fremitus more pronounced at the right than at the left apex. Occasionally the breathing may even be tubular at the extreme right apex. This difference probably depends upon the following facts: the right main bronchus is slightly wider and more vertical in direction than the left; the bronchus to the upper lobe is given off higher up, that is, nearer to the trachea; and the apex of the lung lies slightly closer to the trachea on the right side. The general effect of these conditions is to favour the conduction of the glottic sounds to the right apex. Accordingly we must bear this in mind in estimating the importance of any slight want of symmetry of the auscultatory signs at the apices.

In some instances a slight impairment of the inspiratory expansion of the affected apex and some flattening below the clavicle may be the only physical indications of disease. The want of mobility may be recognised by inspection, but is more easily detected by palpation; the hands of the observer being placed on the subclavian region on each side, and the patient directed to breathe deeply meanwhile. At this time also fine crackling or subcrepitant râles may be heard over the affected area. These râles, which are less fine than the true crepitant râle of Laennec, are mostly heard during inspiration, and are probably caused by the separation of the moist surfaces of the small bronchi. In some cases where the bronchial obstruction is more pronounced no adventitious sounds are audible during ordinary respiration, but when the patient coughs, a shower of crackling râles is produced by the explosive separation of the swollen bronchial walls. At times subcrepitant râles can only be elicited during the deep inspiration that follows cough.

Persistent rhonchi at one apex may sometimes be the only adventitious sounds. A systolic murmur, heard beneath the clavicle, was thought to indicate consolidation of the apex of the lung, and was attributed to the effects of pressure of the infiltrated lung on the subclavian artery; or, with greater probability, to contraction of the thickened pleura at the apex. Similar murmurs may be heard in anæmic and other persons, and are not any certain guide to disease of the lungs. Thus far physical signs give evidence of bronchitis confined to the apex of the lung, the character of the breath-sounds possibly suggesting the existence of a small patch of consolidation surrounded by spongy lung. As the infiltration is often massed at several centres, islets of spongy tissue separate the individual nodules, and for a time mask to a great extent the signs of consolidation. Thus when the lobules around the tuberculous patches are hyper-inflated the percussion may be slightly higher pitched than normal, tympanitic, or even hyper-resonant.

As the disease extends, the lung becomes more airless, and adhesive pleurisy is set up. The inspiratory expansion becomes decidedly restricted, vocal fremitus is increased, and the percussion resonance undergoes progressive impairment. The breath-sounds assume a more definitely tubular or cavernous quality; bronchophony or pectoriloquy appears, and the râles become larger and more ringing or metallic. This complete assemblage of signs is by no means generally, or indeed often presented, except in fairly advanced cases. Tubular breathing may appear at a comparatively early period, but this is unusual; and with marked dulness, bronchophony, and coarse crackling râles, the respiratory murmur may remain simply weakened with slight prolongation of expiration.

The comparatively late appearance of tubular breathing is mainly due to the obstruction of the bronchi, which is so generally present, and to the irregular composite nature of tuberculous consolidation.

Dulness appears first at the supraclavicular and supraspinous fossæ, and thence extends downwards over the front of the chest. For the recognition of slight degrees of dulness light percussion and careful attention to the sense of resistance are required. Increased conduction of the heart's sounds to the corresponding apex generally accompanies and sometimes precedes loss of resonance to percussion. Increasing size of the râles, with a sharply conducted or ringing character, is generally described as marking the presence of softening; but the same signs may be furnished by profusely secreting bronchi of considerable size situated in solid lung.

Rhonchi of a croaking or metallic quality are not uncommon at this period, this peculiarity being imparted to them by the adjacent solid lung or cavities. Dulness gradually extends over a considerable portion of the upper lobe, and râles become audible at the apex of the opposite lung, and at the infraspinous fossa on the same side; that is, at the apex of the lower lobe.

The date at which signs of excavation can be recognised varies greatly. In some cases a cavity may be detected almost as soon as consolidation can be diagnosed, whereas in others it may be months or even years before this is possible.

Signs of excavation.—Over a cavity of considerable size the percussion is generally more or less impaired, and it may be markedly dull in consequence of the surrounding infiltration and of the pleuritic thickening which so often coexist. As excavation proceeds, the dulness may diminish. The resonance is often of tympanitic, tubular, or amphoric quality, as well as slightly dull, resembling the note produced by percussion of the trachea in the neck. Percussion may elicit the cracked pot sound where a cavity communicates freely with the bronchus, and its walls are sufficiently elastic. This sign is not pathognomonic of a cavity, as it may often be obtained by percussing the chest of a healthy infant while crying, and is sometimes found in cases of pneumonia and pleural effusion. The bell sound—"bruit d'airain"—is occasionally heard over large cavities.

According to Wintrich, the pitch of the tympanitic percussion sound over a cavity becomes raised when the patient opens his mouth. An alteration of pitch may sometimes be recognised when the patient changes from the sitting to the lying position, or conversely (Gerhardt); but these changes are seldom pronounced, and give little practical assistance. It is, however, to auscultation that we must mainly trust for the diagnosis of pulmonary excavation. In well-marked cases the breath-sounds are tubular or cavernous—the term "tubular" is used here as synonymous with "bronchial."

Some writers maintain that there is no difference between tubular and cavernous breathing, unless it be in the greater intensity and hollowness of the latter. Flint makes the relative pitch of the inspiratory and expiratory sounds the basis of distinction. According to this author, cavernous breathing is generally of lower pitch than tubular, and the expiratory sound is of lower pitch than the inspiratory; whereas the pitch of tubular breathing is generally higher than that of cavernous respiration, and expiration is higher pitched than inspiration.

The breath-sounds over a cavity may be very weak, or even absent when the bronchial opening is small or obstructed in any way, as by profuse secretion or by cicatricial contraction. If the vomica be separated from the chest wall by a zone of spongy lung the respiration may be simply blowing, with prolonged expiration.

The "metamorphosing" breathing of Seitz consists of an inspiratory sound, harsh or rough at its commencement, becoming hollow or tubular towards the end of the act of inspiration. This sound is supposed to be due to the removal of a partial obstruction of a bronchus as inspiration proceeds. It is not a common sign, and it is not certain that it is exclusively a cavernous sign. Amphoric breathing is pathognomonic of a large air-containing cavity with smooth walls. Large gurgling râles are often heard where cavities contain abundant secretion, and this may be the only auscultatory evidence available at times. When such sounds are audible in regions like the apex, which contains no bronchi of large size, they are very significant of cavities. Auscultation of the cough gives valuable, perhaps the most valuable, evidence of excavation. In a cavity containing fluid and air the agitation produced by cough often gives rise to râles of a splashing character, resembling on a small scale the succussion sound of pneumothorax. Râles of this description are very suggestive of a cavernous origin. A metallic or amphoric echo of the cough is less common but is quite characteristic.

Post-tussic suction is another highly significant sign; it consists of a high-pitched, sucking, inspiratory sound, immediately following the forced expiration of cough, and is due to the elastic recoil of the cavity walls. This has been well named the "india-rubber ball sound" by Dr. Mitchell Bruce.

Metallic tinkling is occasionally heard over large smooth-walled cavities. The vocal resonance is generally increased, bronchophony or pectoriloquy being very common; but the latter is not so decisive a test of excavation as is generally believed. In rare cases an amphoric quality is imparted to the voice when other metallic phenomena are present. In some cavities, where the breath-sounds are feeble, the resonance of the voice may be diminished, especially if the bronchus be obstructed. Cardio-pulmonary systolic murmurs are sometimes heard over large thin-walled superficial cavities lying close to the heart, mostly in the left upper lobe. These murmurs are caused by expulsion of air from the cavity through a bronchus by the impact of the heart on the lung.

Similar cardio-pulmonary murmurs may be occasioned in the absence of any cavity in the lung, if the heart's action be much excited. In cases of contractile disease of the left upper lobe, a systolic murmur is not uncommonly audible in the second left interspace close to the sternum, the bruit being due to traction of the lung on the pulmonary artery. In one case of this sort there was also a marked systolic thrill in this region, which suggested the possibility of stenosis of the pulmonary artery; but an autopsy showed that it was due simply to contracting lung.

A few instances have been recorded in which a systolic murmur was produced by an unsupported and dilated branch of the pulmonary artery crossing a cavity. It should be mentioned that the chest wall may be markedly retracted over the site of a chronic contracting cavity.

Some writers, following Sir Andrew Clark, recognise "fibroid phthisis" as a peculiar variety. Most of these cases are but pulmonary tuberculosis in a very chronic form. There is little in the physical signs to distinguish them from non-tuberculous chronic pneumonia, except that in the former the disease is nearly always most pronounced in the upper lobe, and the apex of the other lung is often involved. In the fibroid or contractile form of pulmonary tuberculosis, signs of excavation are generally to be recognised at one apex, associated with much dulness over the upper lobe or over the whole lung, together with displacement of neighbouring organs. When the left lung is affected the heart is drawn outwards and upwards, and pulsation may be felt as high as the second rib or clavicle; or the apex beat may be discovered in the axilla. In such cases the shock of the second sound may often be recognised in the upper intercostal spaces by palpation. In two of the most extreme instances of displacement of the heart in this disease I found the heart beating under the angle of the left scapula.

When the right lung is contracted the heart is drawn over and may lie wholly to the right of the middle line, the pulsation sometimes reaching as far out as the right axilla. The diaphragm and abdominal viscera are raised by the contracting lung, especially when the upper lobe is principally involved. On the left side the tympanitic resonance of the stomach may extend as high as the fourth or fifth rib. In extreme contractile cases the opposite lung is always considerably enlarged, and may pass beyond the middle line of the sternum into the opposite half of the thoracic cavity. It is often extremely difficult to detect any signs of disease in a lung thus distended, though a post-mortem examination in these circumstances will nearly always reveal the existence of deeply-seated tuberculous lesions. The fact cannot be too strongly insisted upon, that in the presence of distension or emphysema of the lung extensive foci of disease may escape recognition altogether.

Irregular forms.—It seems advisable, at this point, to make a few remarks concerning the physical diagnosis of certain irregular forms of the disease.

Emphysematous form.— In this variety the history as well as the physical signs are those of bronchitis and emphysema. In addition to hyper-resonance on percussion, together with weak inspiratory and prolonged expiratory murmurs, careful percussion will sometimes elicit slight comparative dulness at one supraspinous fossa, and perhaps above the clavicle. There may be no further deviation from the normal type of emphysema. In other cases, on coughing, a few muffled râles may be audible at one apex. If, as often happens, diffused rhonchi are also present, the difficulties of diagnosis are much increased. The shape of the chest is often flat instead of being rounded, a matter of some importance. In emphysematous people with such a formation of thorax, especially if there be much wasting or if hæmoptysis have occurred at any time, the possibility of tuberculosis should be carefully considered, and the sputum should be repeatedly examined for tubercle bacilli.

Pleuritic form.—Reference has already been made to the onset of pulmonary tuberculosis with symptoms of pleurisy. Signs of fluid effusion, thickened pleura, or dry pleurisy in one axilla or at the base, may be the only recognisable signs. It is of the utmost importance in all cases of pleurisy to keep in mind the close relation of this affection to tuberculosis. Double pleurisy, whether there be effusion of fluid or not, is nearly always tuberculous—the principal affections that have to be excluded being renal disease, acute rheumatism, and intra-thoracic growths. Where a large effusion occupies the whole of one pleural cavity, no evidence of tuberculosis can be obtained from physical examination of the affected side.

At times râles or other morbid signs may be detected at the apex of the other lung, but too much importance must not be attributed to such a discovery, as in cases of this description the unaffected lung is often the seat of compensatory hyperæmia and œdema. Similar evidence of apical disease in cases of basic dry pleurisy, on the contrary, has a very definite and positive value. But the sputum may be the only trustworthy evidence of the tuberculous nature of the complaint.

It is commonly said that an insidious onset characterises tuberculous pleurisy, whereas an acute invasion is more suggestive of the simple idiopathic variety. No reliance can be placed on such statements. Tuberculous pleurisy may commence in the most acute manner; and a chronic insidious onset is not rarely witnessed in cases of a comparatively harmless nature. In any case of pleurisy, marked wasting, or a history of hæmoptysis, should arouse suspicion.

Anomalous distribution of physical signs.—When signs of infiltration or excavation are confined to one base, or predominate there, an accurate diagnosis may be very difficult, in view of the extreme rarity of primary tuberculosis of this part. The fact that physical signs of disease are confined to or predominate at the base, by no means proves that there is not, at the same time, older disease of the apex of the upper lobe, a point which I have several times established on post-mortem examination. This depends on the fact that when the lesions are covered by a shell of healthy lung considerable masses of tuberculous disease, or even cavities, may exist towards the central part of the upper lobe without giving any evidence of their presence.

Disease confined to the base of one lung in most cases is not tuberculous, and we have, in such instances, to exclude various affections, the most important of which I may here enumerate:—chronic pneumonia with or without bronchial dilatation, localised pleurisy, abscess of the liver, new growths, hydatid cysts of the lung or liver, and hypophrenic abscess. Examination of the sputum is of the utmost value under such circumstances.

It is well, at the same time, to remember that these diseases may be complicated by a secondary tuberculosis, and the discovery of tubercle bacilli may divert attention from the primary affection. Chronic contracting lesions of the apex of the upper lobe, particularly on the right side, may so uncover the great vessels at the base of the heart as to cause pulsation to be felt in the upper intercostal spaces, and thus aneurysm may be simulated. This is more likely to occur on the right side, where, on more than one occasion, I have known the association of dulness, pulsation, systolic murmur, and accentuated second sound to give rise to considerable suspicion of aortic aneurysm in middle-aged men.

Laryngeal form.—Where laryngeal obstruction exists the entry of air into the lung may be so greatly diminished that auscultation may give no trustworthy indications of the actual condition of the lungs. The amount of pulmonary disease, without any corresponding auscultatory signs, which may exist under such circumstances is surprising, and can only be appreciated by those who have been able to compare the post-mortem appearances with the results of physical examination during the patient's life. Percussion sometimes gives more valuable assistance than auscultation; but the most certain information is often afforded by the sputum test.

Diagnosis.—The diagnosis rests, in the first place, on the presence of chronic disease of the lung, affecting mainly or exclusively the apex of the upper lobe. Signs of persistent catarrh, consolidation, or excavation of this part are, for practical purposes, conclusive evidence of tuberculous disease.

The existence of tubercle bacilli in the sputum is an absolute proof of tuberculosis of some part of the respiratory tract. In the absence of tuberculous ulceration of the larynx, pharynx, or oral cavity, the lung may be regarded as the source of the bacilli, even if auscultation and percussion give no indication of any pulmonary lesion, or if physical signs of disease be found in aberrant situations.

Most writers consider the subject of physical diagnosis under three stages—the first, second and third stages of phthisis. Such a division of the subject implies that physical examination may be trusted to decide at which of these stages the disease has arrived—an assumption by no means warranted by the facts. As a description of the history of individual tuberculous foci, there is not much fault to be found with the time-honoured division into three stages of consolidation, softening, and excavation; but these distinctions are, to some extent, misleading. In the first place, as soon as the stage of softening is reached excavation has begun; in other words, the two processes are, more or less, concomitant; moreover, the rule is to find in the same lung—often in close proximity—solid nodules, softening caseous masses and fully formed cavities: in other words, all three stages are run simultaneously.

On the clinical side of the question auscultation and percussion enable us to recognise consolidation with no little accuracy, and in many cases the existence of a cavity is revealed by certain physical signs; but there is no distinctive sign of softening whatever. In the majority of cases, where, as the result of physical examination, the patient is said to be suffering from phthisis in the first stage, cavities already exist. This is frequently proved to demonstration by the detection of elastic tissue and numerous bacilli in the sputum of cases in which auscultation and percussion point only to catarrh, or to slight consolidation of one apex. It is a matter of everyday experience that cavities in the lung may escape detection during life; and I have known the most experienced physicians diagnose excavation where post-mortem examination showed that none existed. The effects of this artificial classification on the patient's mind have, in many instances, been most pernicious. For, knowing that there are three stages, and hearing that he has a cavity in his lung, he concludes that, as he is in the last stage, his days are numbered. As a matter of fact, many persons in whom a cavity can be diagnosed are in a better condition, and have far more favourable prospects, than others in whom there are only signs of the first stage. It is time that the three stages were consigned to a well-merited oblivion. I make these remarks in no wish to detract from the importance of physical examination; my object is rather to recognise the limitations of this valuable method, and to give a caution against the overweening confidence still reposed by some physicians in auscultation and percussion, to the exclusion of other means of diagnosis.

The complications of phthisis are mostly referable to the transmission of the tubercle bacilli to other parts of the body. In the case of the pharynx, larynx, and trachea, tuberculous changes are mainly produced by the direct inoculation of these parts with the sputum which is constantly passing over them. But in secondary tuberculosis of the genito-urinary, nervous and osseous systems, infection is conveyed by the blood—the microbes, for the most part, effecting an entrance into the circulation through branches of the pulmonary veins.

Laryngeal tuberculosis is almost always secondary to the same disease of the lungs, though in a few well-authenticated cases the lungs have been found on post-mortem examination to be unaffected. The larynx is very frequently implicated; according to my post-mortem statistics this happened in 50 per cent of all cases of pulmonary tuberculosis. In many cases the lesions were recent, and were evidently due to late infection of the larynx. If we exclude all patients in the last stages, it may be said that laryngeal tuberculosis is clinically recognisable in from 20 to 25 per cent. The lesions consist of infiltration or swelling and ulceration. The localisation is a matter of great diagnostic importance.

Tuberculous affections show a marked preference for the posterior part of the larynx, the hinder extremities of the vocal cords, the interarytænoid fold, and the laryngeal surface of the arytænoid cartilages. The epiglottis is less frequently implicated, and the ventricular bands are seldom involved, except in widespread disease of the larynx. The progress of tuberculosis is slow, contrasting strongly with the relatively rapid course of tertiary syphilitic ulceration. The early symptoms are those of chronic laryngitis; hoarseness, tickling, a sense of fatigue on using the voice, and various other paræsthesiæ referred to the throat. Pain on swallowing is a far more important symptom, and is generally associated with swelling or ulceration of the epiglottis or arytænoid regions. Inspiratory stridor and dyspnœa depend for the most part on massive swelling of the epiglottis and aryepiglottic folds; but in certain cases extreme stenosis occurs from mechanical fixation of the cords in the median position, in consequence of infiltration around the crico-arytænoid joints.

In the obstructive form of laryngeal tuberculosis difficulties in physical examination of the chest frequently arise; for when the entry of air into the lungs is much curtailed auscultation may discover nothing more than weakness of the breath-sounds. Hence the importance of an accurate laryngoscopic diagnosis, and repeated examination of the sputum cannot be too strongly insisted upon. For an account of the laryngoscopic appearances, and for further details of this important affection, the reader is referred to the article "Larynx." It should not be forgotten that aphonia in phthisical persons is not uncommonly the result of functional paresis of the adductor muscles of the vocal cords. The trachea is rarely affected except in advanced cases of pulmonary tuberculosis, and the larynx nearly always shows similar and more extensive disease.

Bronchial glands.—The bronchial, mediastinal, and tracheal glands are very prone to tuberculous disease. In adult cases this adenopathy, as the French style it, scarcely ever gives rise to definite symptoms or physical signs. The glands most affected are the anterior or pretracheal, and the subtracheal which lie beneath the fork of the trachea. In children the enlargement of the glands may be so pronounced as to cause obstruction of the large bronchial tubes, or even of the trachea. Bronchial obstruction, if pronounced, leads to pulmonary collapse; in which case dulness on percussion and weakness of the breath-sounds, or tubular breathing, will be found over the affected area. When the upper lobe is concerned the similarity to phthisis may be very close. In some cases the continued absence of adventitious sounds may suggest the glandular origin of the lesion, as in some cases under my care which ended in recovery. Dulness and tracheal breathing over the manubrium may occasionally be found when the pretracheal glands are greatly enlarged. It is said that dulness may be recognised in the upper interscapular region; but I have never met with this myself; and it seems unlikely that enlarged glands in the fork of the trachea and, therefore, lying in front of the spine, should occasion dulness in the situation indicated. The subjects of this complaint sometimes suffer from a spasmodic cough like whooping-cough, and from attacks of dyspnœa, attributable to pressure on the vagus trunks.

Compression of the recurrent laryngeal nerve, more particularly on the left side, may cause paralysis of the corresponding vocal cord. Perforation of the œsophagus by a suppurating caseous gland, when the abscess opens into a bronchus, is apt to give rise to septic bronchopneumonia, and gangrene of the lung may follow. Rupture of a glandular abscess into the trachea may cause fatal asphyxia. In many instances caseous glands undergo calcification, and the disease is thus arrested.

Pneumothorax is one of the most serious and fatal complications, statistics proving that the patient rarely survives this accident by more than one month at most; though exceptions to this rule are to be met with. It is at first sight remarkable that pneumothorax does not occur more frequently, considering the tendency of pulmonary cavities to extend outwards towards the pleura. Dr. Samuel West's experiments enable us to understand why perforation of the visceral pleura is not necessarily followed by pneumothorax, even when there are no adhesions. For, as he shows, before the elastic recoil of the lung can assert itself, the normal cohesion of the two layers of the pleura must be overcome, and this requires considerable force; in other words, the force of cohesion considerably exceeds the elasticity of the lungs. He concludes that "pneumothorax, in its initial stage, must be an active process. Some force will be required to overcome the normal cohesion between the two layers of the pleura, and to separate them. This must be obtained by expiration, and pneumothorax, therefore, in its initial stage, is an expiratory process, and not essentially different in its production from surgical emphysema. As soon, however, as separation has been effected, the elasticity of the lungs will come into play, and air will enter the pleura until its retractility is completely satisfied" (*vide* p. 335).

Inasmuch as perforation of the pleura is always succeeded by inflammation the force of cohesion may soon be supplemented by adhesive pleurisy, and the entry of air into the pleural sac may be thus prevented. In cases where the opposite lung is extensively diseased the dyspnœa at first is very great, and death may occur in a few minutes; but the immediate consequences of the perforation are almost invariably recovered from. Physical examination on the affected side shows absence of movement, increased fulness of the intercostal spaces, diminished tactile fremitus, and hyper-resonance or tympanitic percussion note. On auscultation the breath-sounds are absent or feeble—at times amphoric, and the vocal resonance is diminished; occasionally amphoric echo of the voice may be obtained. Percussion by means of coins, or with a pleximeter and percussion hammer, while the stethoscope or naked ear is applied to the chest, yields a clear metallic sound, the bell sound, or *bruit d'airain*. Metallic tinkling and amphoric echo of the cough may also be heard. The hippocratic succussion splash can often be detected when fluid effusion has occurred, if the ear be placed on the chest and the patient be shaken sharply. The heart is displaced to the opposite side, except in the rare instances where it is fixed to the sternum by adhesions, or where the opposite lung is solidified or completely adherent.

This displacement is not due to the pressure of the pneumothorax as is commonly assumed; for in such cases, as shown by Sir R. Douglas Powell, manometric measurements may indicate no positive pressure in the pleural cavity; and his experiments have demonstrated that the dislocation of the heart is due to the unopposed elastic traction of the sound lung. The diaphragm and the abdominal viscera on the corresponding side, being no longer held up by the elasticity of the lung, sink downwards. In some cases depression of the liver or spleen may be detected by palpation. Although effusion nearly always ensues, it may be difficult to obtain clear evidence of its presence. Sometimes there is a small area of dulness at the base, shifting, to an unusual degree, with the position of the patient. In other cases there may be no signs of fluid except the succussion splash, which, however, is quite decisive. The absence of dulness is to be explained by collection of the fluid in the cup-shaped space formed by the depressed diaphragm. In more chronic pneumothorax a copious exudation may occur, and the air gradually become absorbed. Under these circumstances there will be marked dulness and other signs of simple pleural effusion, from which the case can only be distinguished by the history. The effused fluid is generally purulent, but may be sero-fibrinous.

Instances of complete recovery after pneumothorax have been recorded by many observers. In most of these the perforation of the pleura occurred without any previous evidence of pulmonary disease; and, although it is probable that many of them were tuberculous, this cannot be stated with certainty. In a much smaller number of cases, where pneumothorax appeared in the course of manifest pulmonary disease, life has been prolonged for months or years. The occurrence of pneumothorax seems, in some instances, to exercise an inhibitory effect on the disease in the affected lung—a result probably to be attributed to the diminished blood-supply consequent on the pulmonary collapse.

Pleurisy.—A certain degree of pleurisy occurs in every case, although it may be unaccompanied by any symptoms. Signs of dry pleurisy, without any evidence of effusion, are often met with. When a dry rub is heard over a considerable area—usually the lower part of the chest—it not uncommonly indicates progressive disease; but there are many exceptions to this rule. Pleural effusion occurring in the course of pronounced phthisis is seldom very profuse, perhaps because the pleural cavity has been already partly obliterated by adhesions. The fluid is generally sero-fibrinous, sometimes purulent, and occasionally sanguineous. Cases have been recorded where rapid absorption of an effusion was followed by acute generalised tuberculosis. This, however, is a very rare sequence of events, and the relation may be accidental. Some cases of tuberculous empyema have originated in pneumothorax, where the opening has been closed by inflammation, and the air has been gradually absorbed. Empyema is much more unfavourable than sero-fibrinous effusion, as absorption cannot be expected, and treatment by incision is rarely successful. Small empyemas very occasionally undergo inspissation and arrest. Sanguineous effusion is less common than the statements of writers would lead one to suppose. Pleural effusion, like pneumothorax, exercises a retarding influence on the pulmonary disease in virtue of the collapse of the lung which ensues.

Pneumonia.—As already stated in the section on Pathology, croupous pneumonia occasionally attacks phthisical patients; but this is very uncommon. Most of the authors who mention this subject consider that the course of phthisis is not materially influenced by intercurrent pneumonia. In the only instance of this accident that I have met with, the pneumonia ended favourably with a well-defined crisis, and the old apex lesion was left in the same condition as before the acute attack.

Tuberculous persons are apt to acquire more or less acute bronchopneumonia from time to time; but most of these attacks represent acute exacerbations of the tuberculous process. Influenza, attacking the subjects of phthisis, may set up pneumonia of the broncho-pneumonic kind, less frequently the lobar.

Circulatory system. — The heart of phthisical persons is small, and shows atrophic changes, occasionally slight fatty degeneration, and very rarely solitary tuberculous masses in its muscular walls. It is rarely that such lesions give rise to any functional disturbance. In some of the most chronic cases dilatation of the right ventricle may occur.

Endocarditis is not very uncommon, and is sometimes attributable to previous attacks of acute rheumatism, but by no means always. Some French observers state that they have discovered tubercle bacilli in the valvular vegetations in such cases; but the relation of endocarditis to tuberculosis is still in need of investigation. Dilatation of the heart, whether due to valvular defects or myocardial disease, exercises a retarding effect on the progress of pulmonary tuberculosis. Attacks resembling pseudo-angina pectoris may be encountered; and it is said that they occur more often where the left upper lobe is contracted and the heart much exposed. It is doubtful whether this association amounts to anything more than a coïncidence.

Pericarditis is generally due to extension of tuberculosis from the pleura or anterior mediastinal glands, or occasionally from the peritoneum. In a few recorded cases a pulmonary cavity has perforated the pericardium, and produced pyopneumopericardium. Tuberculous granulations or caseous nodules may be seen in the serous membrane; or the tuberculous nature of the affection may only be demonstrable by the microscope. The effusion, as a rule, is scanty and sero-fibrinous in character; occasionally purulent or hæmorrhagic. There is always much fibrinous exudation, and usually more or less adhesion of the two layers. Tuberculous pericarditis generally escapes recognition during the patient's life; though, from its weakening effect on the muscular wall of the heart, it must be regarded as an important complication.

Pulmonary embolism, from detachment of thrombi formed in the right ventricle or auricle, is an occasional occurrence. When hæmorrhagic infarction of the lungs ensues the condition may generally be diagnosed. But if no infarction be produced embolism may pass unrecognised, especially in moribund patients. Thrombosis of branches of the pulmonary artery may take place in the last stages, but this is not a common event. In some advanced cases we find great œdema of one leg from thrombosis of the large veins. Tenderness and induration can generally be discovered in the course of the affected vessel. Purpuric spots may appear on the lower extremities in conditions of cardiac debility.

Alimentary canal.—Tuberculous ulceration of the lip is extremely rare, but the tongue and other parts of the oral cavity are more often affected. Ulceration of the tongue appears most commonly on the dorsum, but it may attack the sides, and occasionally the frænum. In cases of extensive tuberculosis of the soft palate and pharynx ulceration sometimes invades the buccal mucous membrane and the gums. The soft palate, uvula, and the pillars of the fauces are more often attacked; the prevailing lesion consisting of diffuse submucous infiltration and swelling, with shallow serpiginous ulceration. Miliary nodules may be seen in the base of the ulcer at times. Tuberculosis attacks the posterior wall of the pharynx less frequently than the palate. The usual lesions are circular ulcers with raised edges and granulations in the base, and superficial ulceration extending from the posterior pillars of the fauces. In some instances the larynx also is extensively affected, and the tuberculous disease appears to have originated there. But ulceration of the pharynx or tongue may occur without any laryngeal complication, and is generally due to infection from the sputum; but it may be part of a generalised tuberculosis.

Tuberculous ulceration of the oral cavity may be occasionally mistaken for syphilis, or for malignant disease. Herpes of the pharynx simulated miliary tuberculosis of the soft palate for a time in two tuberculous patients who came under my notice. For the diagnosis of such cases reference should be made to the article "Pharynx" (vol. iv. p. 745). In tuberculous affections of these parts pain is always a prominent symptom, and interferes greatly with the act of deglutition; in consequence of which the nutrition of the patient suffers seriously. Aphthous stomatitis is a fairly common complication in the terminal stages, and may occasion great discomfort.

The tongue presents no special features in phthisical patients, and its condition varies with the state of the oral cavity and alimentary canal. In cases of intestinal ulceration it is sometimes red, glazed and raw-looking; but similar appearances may be observed where no ulceration of the stomach or intestine exists. The red line on the gums, to which much attention was paid formerly, is by no means characteristic, and, moreover, is not very frequent.

Isolated instances of œsophageal tuberculosis have been recorded, but the gullet rarely shows any morbid change. Tuberculous ulceration of the stomach is extremely rare. A mammillated condition, pointing to chronic gastritis, is not uncommon. Chronic interstitial gastritis, atrophy of the glandular cells, and dilatation of the stomach have been found in some cases, but, as a rule, no morbid appearances are presented; the gastric symptoms are mostly dependent on functional derangements. Symptoms of dyspepsia, such as loss of appetite, cardialgia, flatulence, and constipation, are very common. Vomiting is often a very troublesome symptom: sometimes it is associated with a red irritable state of the tongue and epigastric pain, and is attributable to gastric catarrh; but more frequently it is unrelated to any affection of the stomach, and is excited by fits of coughing, which are apt to arise after meals and are possibly a result of hyperæsthesia of the vagus. Attention to the state of the stomach and digestion is of great importance in the treatment of all cases.

The *intestine* is more often the seat of secondary tuberculosis than any other organ. In my post-mortem examinations the intestine was involved in 70 per cent of all cases of phthisis. The lesions are mostly situated close to the ileo-cæcal valve; the last few feet of the ileum, and the cæcum being most frequently attacked: but tuberculosis may show itself in any part of the alimentary canal from the duodenum to the anus. The fact that the process begins in Peyer's patches and the solitary follicles, where the lymphatic system is most highly developed, suggests that the virus is absorbed from the intestine; and it is probable that the bacilli are conveyed by sputum, which has been swallowed.

In the small intestine the ulcers are at first more or less rounded, and extend laterally, the edges and base being thickened, and the latter often studded with granulations or small caseous foci. On the peritoneal surface groups of miliary tubercles are often seen, with localised peritonitis; and on this surface whitish beaded cords, representing lymphatics filled with tuberculous material, may be traced from the ulcer towards the mesentery. In the colon the ulcers are more elongated in a transverse direction, and often partially or wholly encircle the gut. Thickening is less conspicuous than in ulceration of the small intestine, and subserous tubercles and localised peritonitis are seldom seen. Partial cicatrisation of tuberculous ulcers is not uncommon, and at times stenosis may result. Owing to the thickening of the base of the ulcers, and the marked tendency to the formation of adhesions between neighbouring coils of intestine, perforation is generally prevented; but this accident is less rare than is generally supposed: the peritonitis which ensues will be restricted or general according to the presence or absence of adhesions. Circumscribed purulent peritonitis is by no means rare; and, when occurring in the cæcal region, is very liable to be mistaken for simple perityphlitis. The symptoms of intestinal tuberculosis are few and uncertain; they may be indicated as diarrhœa, localised pain and tenderness in the abdomen; but, unfortunately, none of these can be depended upon. Cases of the most severe ulceration of the small intestine or colon may run their course without any definite pain or tenderness, and may be accompanied by obstinate constipation from paralysis of the muscular fibres of the gut. Diarrhœa may be due to other causes, especially enteric catarrh and lardaceous disease. In the case of ulceration the stools may have a pale yellow or drab colour, but they commonly present no characteristic features. Local tenderness is more common with the diarrhœa of ulceration. In some instances the discovery of tubercle bacilli in the motions will put the diagnosis beyond all doubt. The presence of pus in the stools cannot often be detected, and is generally symptomatic of ulceration, in which case bacilli are likely to be found; but an abscess communicating with the intestine will have to be excluded: a large amount of pus would be in favour of an abscess. Small quantities of blood may be discharged with the motions, but copious hæmorrhage is very rare: however, in two patients under my care death resulted from profuse bleeding. In one case only could a post-mortem examination be obtained, and here a tuberculous ulcer of the colon was found to be the cause of the hæmorrhage. In severe cases of ulceration the activity of the process in the lungs seems, at times, to become arrested.

Fistula in ano can sometimes be traced to a burrowing tuberculous ulcer of the rectum; but it is not uncommon, in cases of this description, to find the lower part of the bowel free from ulceration or obvious disease.

It is by no means certain that ischio-rectal abscess is always or indeed generally of tuberculous origin.

In two female patients who came under my observation, with advanced tuberculous ulceration of the intestine and rectum, the muco-cutaneous margin of the anus and the neighbouring skin were affected with a superficial serpiginous ulceration of similar nature.

The diagnosis of lardaceous disease of the intestine can only be arrived at when there are signs of similar disease of the liver, spleen, or kidney. Enlargement of the spleen or liver, with albuminuria, casts in the urine and polyuria, coexisting with diarrhœa, would strongly suggest lardaceous disease; but it must be remembered that lardaceous degeneration and tuberculous ulceration may exist in the same patient and in the same intestine. A marked degree of anæmia is very general in cases of lardaceous degeneration. Transient diarrhœa is mostly attributable to simple catarrh, the diarrhœa of ulceration and amyloid disease being very persistent.

The liver may contain miliary tubercles, large caseous nodules, and occasionally tubercular abscesses; but, as a rule, these affections are clinically unrecognisable. In one case that I examined a hypophrenic abscess was caused by a perforating tuberculous abscess of the left lobe of the liver. Enlargement of the organ is most frequently caused by fatty and amyloid degeneration. The presence of a large spleen, albuminuria, and diarrhœa would be in favour of lardaceous disease, especially if the edge of the liver be thick and very firm. Cirrhotic enlargement is relatively of frequent occurrence in cases of chronic tuberculous peritonitis. It is possible that cirrhosis may be causally related to peritoneal tuberculosis. Miliary tubercles and extensive fatty degeneration are commonly associated with the cirrhosis of tuberculous subjects.

Enlargement of the spleen is a frequent symptom of lardaceous disease, and is only likely to be confounded with the secondary splenic tumour of hepatic cirrhosis. In both cases the spleen is very firm. In acute generalised tuberculosis, as in other specific fevers, the spleen may be enlarged, whether it contain miliary tubercles or not; but its consistency is soft. Caseous nodules are often found in the spleen, especially in children, but they possess no clinical importance.

Tuberculous peritonitis may be part of a general tuberculosis, or it may be due to extension from the abdominal organs—intestine, lymphatic glands, and female generative organs; or it may be the result of infection from the pleura or pericardium, the bacilli being transmitted through the lymph spaces of the diaphragm.

Miliary tuberculosis of the peritoneum is often unaccompanied by any symptom whatever; but it may give rise to ascites, in which case some degree of chronic peritonitis will be found. In another form the tuberculous lesions consist of large nodules or masses, which are generally more or less caseous, but may at times be mainly or entirely fibroid. Caseous and fibro-caseous nodules may coexist in the same case. When the individual nodules coalesce large masses are formed which may be recognised by palpation during life. The great omentum is frequently much thickened, shortened and rolled up, forming a thick transverse band just above the umbilicus; but omental growths may be situated in the lower part of the abdomen also. The omentum may also undergo a general tuberculous infiltration, giving it the appearance of a thick apron hanging down in front of the intestine.

Retraction of the thickened mesentery, fixing the intestine against the spine, sometimes gives the appearance of a tumour. Large tuberculous masses may be found in the pelvis or in any part of the abdomen. When the pelvis is involved the disease has commonly originated in the female generative organs, the bacilli passing from the Fallopian tubes into the peritoneal cavity; but, at other times, the pelvic peritonitis may be secondary to disease in the upper part of the abdomen, the virus having apparently gravitated to the pelvis. In these cases the intestines are always much matted together, and patches of soft lymph, with crops of miliary tubercles, may be found, showing that the disease is still in progress. The fluid exudation is often purulent, but it may be sero-fibrinous or sanguineous. Softening of the caseous masses sometimes leads to perforation of the hollow viscera—intestine or bladder.

Partial or complete arrest of tuberculous peritonitis is by no means rare. Where cicatrisation takes place contraction may ensue and cause stricture of the intestine.

In cases of peritoneal tuberculosis the mesenteric and other lymphatic glands of the abdomen are always more or less enlarged and caseous. The glands may be the only abdominal organs affected, but intestinal lesions are very frequently present. Enlargement of these glands in adults is seldom so extensive as to admit of their being felt through the abdominal walls; but I have known caseous glands in the iliac and umbilical regions to form tumours as large as an orange. The tuberculous glands of children attain to a relatively larger size, and are more often susceptible of palpation; but even in children, and still more in adults, it may be difficult during life to decide whether a tumour be glandular or omental; though a deep situation and greater fixation of the tumour would be rather in favour of the former. The designation "tabes mesenterica" has been shown by Dr. Gairdner and others to comprehend not only tuberculosis of the mesenteric glands, but also tubercular peritonitis and other morbid conditions associated with wasting; and the name has consequently fallen into disuse.

Urogenital system.—Miliary tubercles and small tuberculous foci in the kidney may be accompanied by slight albuminuria, or may cause no symptoms. In the important variety known as scrofulous or tuberculous pyelitis, large areas of the kidney undergo caseous necrosis, and in most instances softening and ulceration ultimately ensue. Inasmuch as the process predominantly involves the pyramids and calices, disintegrated tuberculous material and pus are discharged with the urine from time to time. Tuberculous infiltration may ultimately involve the whole kidney, which then generally becomes enlarged, and may be converted into a loculated thick-walled cyst, containing soft putty-like caseous material; the dilatation of the pelvis being attributable to obstruction to the flow of urine. Both kidneys are affected as a rule, though one is usually in a more advanced stage of the disease. The infiltration and ulceration may extend from the pelvis of the kidney to the ureter, and thence to the bladder, prostate, vesiculæ seminales, vas deferens and epididymis.

The symptoms of this form of renal tuberculosis are mainly the result of the pyelitis which constitutes the most salient feature of the affection —lumbar pain, mostly of dull character, but at times paroxysmal and colicky, when the ureter becomes obstructed, pus with a corresponding quantity of albumin, caseous débris, renal epithelium, and, at times, blood in the urine. Tubercle bacilli may be recognised in the urinary sediment, and are a conclusive proof of tuberculosis. If tuberculous disease of the bladder, prostate, and vesiculæ seminales can be excluded, the existence of renal tuberculosis would amount to a certainty. Occasionally a definite renal tumour can be made out by palpation, but this is the exception.

In addition to the foregoing affection phthisical patients may acquire acute or chronic nephritis, lardaceous disease, and granular kidney. The commonest of these lesions is lardaceous disease. Slight degrees of this degeneration may need the application of iodine for their recognition, and in such cases no clinical symptoms would be presented. The higher grades of this disease are always combined with a varying amount of chronic nephritis, the kidneys in such cases being large, pale, and translucent, with yellowish opaque patches in the cortex. The surface is generally uneven, and the capsule adherent.

The amyloid disease affects principally the glomerular capillaries, but also the small arteries, the vasa afferentia and vasa recta. Degenerative changes in the convoluted tubes are due partly to the obstructive effects of the lardaceous disease of the vessels supplying these structures; and partly to the blood state, in which the lardaceous degeneration itself originated. In association with these changes a varying amount of scattered cell infiltration and fibrosis is nearly always found; these represent reactive inflammation secondary to parenchymatous degeneration. The urine in such cases is abundant, of low density, and contains albumin in considerable quantities, and hyaline casts. Dropsy is uncommon. The other forms of renal disease mentioned above present no features to distinguish them from similar affections in non-tuberculous subjects. Acute nephritis is uncommon, and is probably of hæmatogenous origin and attributable to absorption from ulcerative cavities in the lungs. Granular kidney is not uncommonly met with in elderly and middle-aged persons, and is sometimes accompanied by slight degrees of lardaceous degeneration. It is very doubtful whether there be any causal relation between granular kidney and pulmonary tuberculosis. In cases where albuminuria supervenes a fall of temperature and a diminution of the activity of the pulmonary disease are not uncommonly observed.

Phosphaturia is said by Sir R. Douglas Powell to be an early indication of phthisis. Ehrlich's diazo reaction is found in febrile progressive forms of tuberculosis, but no diagnostic significance can be attached to it. Tuberculous ulceration of the bladder is not very common, and is mostly associated with similar disease of other parts of the genito-urinary system. The symptoms are those of cystitis. Tubercle bacilli may be found in the urine. Tuberculous disease of the epididymis is much less uncommon, but this affection and tuberculosis of the prostate and vesiculæ seminales come rather within the sphere of the surgeon.

Tuberculosis of the uterus is decidedly rare. The disease, which attacks the lining membrane of the fundus, consists of tubercular infiltration, which is soon succeeded by caseous necrosis and ulceration. The uterine cavity commonly contains thick cheesy pus, and is apt to be somewhat dilated. There is rarely much enlargement of the organ. The Fallopian tubes are much more frequently attacked, and are seldom spared where the uterus is affected. In tuberculous salpingitis similar lesions are found in the mucous membrane; but the thickening and dilatation of the tubes attain to much greater proportions.

Tuberculosis of the ovary is one of the rarest occurrences: the only case I have seen is recorded by Dr. Habershon. In this case both ovaries contained tuberculous abscesses which communicated with the Fallopian tubes and intestine.

Tuberculous peritonitis is not uncommonly attributable to extension from the Fallopian tubes or uterus. It is probable that genital tuberculosis may also be caused by infection from the peritoneum; but more often the disease is communicated through the blood. The possibility of direct sexual infection cannot be denied.

Some writers have contended very strongly that pregnancy exercises a retarding influence on the disease; others hold that phthisis is aggravated by this condition: on the whole, pregnancy seems more often to intensify the symptoms of pulmonary tuberculosis. There is little doubt as to the injurious effects of parturition. It is a common experience that after confinement the pulmonary disease makes rapid progress, and is apt to terminate fatally in a few months. Hanau believes that this is to be explained by the inhalation of infective material from cavities into healthy parts of the lungs during the forcible inspirations that accompany expulsion of the fœtus. The exhausting influence of lactation is notorious. Menstruation is nearly always much deranged, apart from any definite lesion of the generative organs. Amenorrhœa, or scanty, infrequent menstruation, is the rule in this disease, and may be one of the earliest symptoms of it. Very occasionally menorrhagia occurs, but is seldom persistent.

The *suprarenal bodies* occasionally contain isolated caseous nodules, which cause no symptoms. Still more rarely both adrenals are converted into firm caseous or caseo-calcareous masses, in which case bronzing of the skin and other symptoms of Addison's disease supervene.

Osseous system.—Secondary tuberculosis of the osseous system and joints is not very common, and may show itself, among other places, in the vertebræ, sternum, and ribs, giving rise to chronic abscess in connection with the chest walls. This subject possesses more surgical than medical interest.

Nervous system.—The mental attitude of many phthisical patients is one of irrepressible hope, especially in the less chronic forms. Such persons often asseverate that if they could but get rid of some particular symptom, such as cough or shortness of breath, they would be perfectly well; and they go on making plans for the future within a few hours of their death. But in most cases presenting definite symptoms of mental derangement depression is the prevailing feature. Melancholia, stupor, delusions of suspicion or persecution, religious foreboding, insomnia, hallucinations, a suicidal tendency, and refusal of food are among the commonest symptoms. Maniacal excitement is much less frequent. For further information the reader is referred to the section on Insanity in the last volume of this work.

Tuberculosis is much less liable to affect the nervous system in the course of chronic phthisis than in acute tuberculosis. It is also of much more frequent occurrence in children than in adults. In most cases the tubercle bacilli are conveyed through the blood. The cerebro-spinal meninges are the parts most commonly attacked, the tuberculous process being grouped especially along the small vessels. The growth of tubercles is soon followed by fibrinous exudation, in consequence of which the pia mater becomes much thickened.

Meningitis nearly always predominates, or is exclusively localised at the base of the brain, and extends thence to the Sylvian fissures, the ventricles, the surface of the cerebellum, the pons Varolii, and the medulla. The ventricles are often much dilated and filled with turbid fluid—"acute hydrocephalus" of the old writers, the convolutions becoming flattened by pressure. The cortex of the brain and the walls of the ventricles are often much softened, from extension of the inflammation of the pia mater, so that the process is more correctly described as a meningo-encephalitis. Tuberculous nodules or masses may grow in the brain tissue, and sometimes attain to a considerable size. These solitary tubercles or tuberculous tumours are found most frequently in the cerebellum and cerebral hemispheres, but they may arise in any part of the brain and are often multiple. Small tuberculous nodules are not infrequently found in the cortex, extending inwards from areas of chronic tuberculous meningitis.

Lastly, meningitis, encephalitis and myelitis may be due to extension from neighbouring bones of the cranium or spine.

The symptoms of meningitis are many, and can only be briefly enumerated:—headache, irritability of temper, fretfulness, coma, convulsions, marked retardation, acceleration, or irregularity of the pulse, Cheyne-Stokes respiration, vomiting, retraction of the head and abdomen, rigidity and weakness of limbs, paralysis of cranial nerves, optic neuritis. Retention of urine is very common towards the close, and pyrexia is nearly always present. Headache is perhaps the most common symptom in the more chronic form. Tuberculous tumours of the brain give rise to symptoms not differing from those of other cerebral tumours. For a full account of this subject reference must be made to the appropriate articles.

Peripheral neuritis has been observed in a small number of cases in the form of extensor paralysis of the arms or legs. The cause of the neuritis is uncertain. It may be the result of toxines, elaborated by the tubercle bacilli.

Some of the pains and tenderness affecting the limbs in phthisical patients may possibly be of neuritic origin. Beau grouped these together under the name "melalgia." It is difficult at present to discriminate the pains which many patients in advanced phthisis complain of. Some are probably neuritic, others myalgic; while, in some instances where pains fly about from one part to another and affect the joints, the resemblance to rheumatism is very close. In these last the rheumatoid pains are possibly a septicæmic symptom, depending on absorption from pus-secreting cavities in the lung. Suppurative otitis media is not very uncommon, but it is seldom that tubercle bacilli can be discovered in the pus.

Course.—The course of pulmonary tuberculosis is essentially variable and fluctuating, intervals of quiescence or apparent arrest alternating with prolonged periods of fever and other constitutional symptoms. In a large percentage of cases the disease is for the most part slowly progressive, and death ensues in a few years at the latest. The average duration of phthisis has been variously estimated. Louis found that in more than half the cases observed death occurred in less than nine months. The mean duration has thus been stated:—twenty-three months (Louis and Bayle); two years (Laennec and Andral); four years (C. J. B. Williams and Sir J. Clark): Dr. C. T. Williams, from analysis of a thousand cases among private patients, put it at seven years and three-quarters. On account of the great difficulty so frequently met with in attempting to fix the date of onset of the disease such calculations are fraught with uncertainty. Those physicians who have had much experience of the disease at special as well as general hospitals, will probably agree that statistics derived from the latter source exclusively would give a very erroneous impression of the duration of phthisis. Patients admitted into general hospitals are either exceptionally ill, or are suffering from some serious complication. The mortality among such patients is naturally very high, and the duration of the disease may often be measured rather by months. Most valuable are the statistics collected by Dr. J. E. Pollock from 3500 cases of phthisis attending the out-patient department of the Brompton Hospital.

"Here (among the out-patients)," as he truly says, "are seen individuals of all classes, excepting the highest, and of all ages and occupations. The necessities of home cares and of continuing the daily work are but little interfered with by a visit once a fortnight to their physician; but these urgent claims of domestic life shut out large numbers from the possibility of availing themselves of indoor treatment in a hospital. The large class affected with chronic slow phthisis are, therefore, found chiefly among the out-patients.

"The average duration, while under observation, of all the cases taken together was two years six months and three-fifths nearly, but this represents only a part of the period of the affection, and in it are included cases of the most acute and rapid form as well as those which have become chronic."

The actual duration of the cases must have been considerably longer, and the whole average duration of the disease, as Dr. Pollock says, must be raised beyond four years. An experience of twelve years' out-patient work at the Brompton Hospital has convinced me that Dr. Pollock is far nearer the mark than those who would limit the average duration to two years.

The complexion of the malady, while running a chronic or slowly advancing course, is liable at any time to undergo a complete change, depending on acute exacerbation of the pulmonary disease. Fever and other constitutional symptoms often herald renewed activity of the tuberculous process before physical examination gives any decided indication of extension. In other instances we find the signs of disease slowly extending for some time without any corresponding aggravation of the patient's symptoms.

The lines along which the disease spreads in the lungs have been described in the section on the pathology. It is very important not to be satisfied with exploration of the front of the chest only, but to examine with care the back also, more especially the supraspinous fossa—that is, the posterior aspect of the upper lobe—and the interscapular region just below the spine of the scapula, which corresponds to the apex of the lower lobe, a part specially prone to secondary tuberculosis. And, as Dr. J. K. Fowler quite rightly insists, search should be made for signs of disease extending from behind forwards from the apex of the lower lobe along the upper border of the same lobe, the position of the septum dividing the upper and lower lobes being roughly indicated by the "vertebral border of the scapula, when, with the hand upon the spine of the opposite scapula, the elbow is raised above the level of the shoulder."

The upper part of the axilla is another region that must be carefully investigated, as it is in this space alone that the outer aspect of the upper lobe is accessible to examination; and signs of excavation may sometimes be found at the apex of the axilla only.

Towards the close of life bubbling râles are generally heard over the whole of the chest, and are an indication of pulmonary œdema, the result of cardiac failure. It is usual to find resonance to percussion over the lower part of one or both lower lobes up to the very end; a fact which is to be explained by the persistence of patches of spongy lung between the tuberculous masses.

Where the fatal termination is not directly or indirectly dependent on complications, but is the result of slowly extending disease, death most frequently occurs from exhaustion. Asphyxia is seldom the cause of death except in acute forms of tuberculosis. In most chronic cases death is preceded by profound emaciation and debility, which steadily increase in spite of the considerable quantity of nourishment the patients often continue to take. Bed-sores may form if the nursing be not vigilant, and œdema of the legs is not uncommon. The pulse becomes more rapid and feeble, the temperature gradually falling often becomes subnormal, tracheal râles appear, and the end comes quite peacefully. In the comparatively few cases in which complete arrest of the disease takes place, the constitutional and local symptoms gradually subside, and the patient regains his health. The physical signs at the same time undergo certain modifications, or occasionally disappear entirely. In most cases, although râles and other adventitious sounds cease to be heard, signs of consolidation and contraction of the apex persist, and some degree of localised emphysema is often developed.

Prognosis.—Of the many complicated problems presented to the physician the prognosis of pulmonary tuberculosis is one of the most difficult. An accurate prognosis would involve full knowledge of the parasite and its host, as well as of their environment. At present little is known concerning variations in the virulence of the tubercle bacillus as it occurs in the body of man.

Still less information is forthcoming as to the histo-chemical and biological conditions of the human organism which retard or favour the development and activity of the parasite. Certain facts concerning the external conditions that appear to exert a salutary or injurious influence on the disease have been discussed under its causation. A complete understanding of these points is intimately connected with the question of immunity, a most difficult subject, which is only just beginning to be studied. For practical purposes we have to estimate the prognosis, in the first instance, by a careful consideration of the effects of the disease, immediate and remote, in each patient. By these means we are able to gauge, approximately, the severity of the malady and the resisting power of the individual. Furthermore, an acquaintance with the natural history of tuberculosis, including the influence of heredity, of previous or concurrent diseases, and of various conditions of life, and lastly, the knowledge of the effects of treatment, will be required if we would forecast the probable course of pulmonary consumption. The symptoms of the patient, representing the result of disordered function, are of the first importance. Of all the general symptoms fever is the most important. A markedly intermittent or remittent pyrexia, in the absence of acute intercurrent affections, is very significant of progressive disease, and is, therefore, of bad augury. At the same time it must be remembered that a considerable degree of fever is not incompatible with gain of weight and other signs of improvement. Moreover, after periods of severe pyrexia the temperature may fall, and the disease enter upon a chronic phase. Nevertheless, it may be accepted as a general principle that the existence of marked pyrexia always necessitates a very guarded, though not necessarily an entirely unfavourable prognosis.

A slight evening rise of temperature, with a fall to normal or slightly below normal in the morning, is not uncommonly present in comparatively favourable cases. The supervention of fever in the course of a mild chronic case is often one of the first indications of renewed activity of the tuberculous process, which may prove intractable. The absence of fever does not in itself justify the expression of a hopeful opinion, for, as we have seen, an apyrexial temperature may accompany advanced and active disease. Subnormal or collapse temperatures have a very ominous import. Emaciation signifies deficient alimentation (whether due to insufficient feeding, digestion, or assimilation), or profound constitutional intoxication. In the first case the cause is more amenable to treatment, and the outlook is consequently less unfavourable. A persistently rapid or easily excited pulse is indicative of debility, or of a state of general nervous erethism, both of which are very undesirable features. Anæmia and debility are also an evidence of profound constitutional impression, and must therefore darken the prognosis.

Among the more important symptoms of local disorder we must reckon dyspnœa depending on diffuse or acutely extending pulmonary changes. When these changes consist in disseminated miliary tubercles, or in lesions of the broncho-pneumonic or pneumonic type, the gravity of the symptom can hardly be exaggerated. Expectoration, profuse, purulent, and containing numerous elastic fibres, implies progressive destruction of lung. Absence or scantiness of expectoration is, at times, a marked feature in severe cases: this is mostly, but not exclusively, seen in children and women, who often swallow their sputum. But while no great importance can be assigned to the quantity of the sputum, scanty or moderate expectoration is on the whole a good sign.

The expectoration of pulmonary calculi is never met with except in very chronic cases.

The significance of fœtor varies with its cause. When the odour has a sickly or slightly fishy character, due to the retention of secretion in cavities, it is of less moment than when it possesses the penetrating odour of bronchiectasis: in the latter case the dangers of septic broncho-pneumonia and other accidents are added to those already existing. The supervention of gangrene renders the prognosis quite hopeless.

The number of tubercle bacilli in the sputum is no accurate measure of the extent or severity of the disease, and is largely a question of discharge. In some acute cases the bacilli may be very scanty, whereas in other cases, quiescent and circumscribed, the sputum may teem with them; complete and permanent disappearance of the microbes is a most hopeful sign; but their continued presence in the sputum does not preclude a protracted and favourable course.

An incessant and intractable cough, especially when it interferes with sleep and causes vomiting, adds greatly to the exhaustion of the patient. Some of the most irritable coughs depend on catarrhal affections of the upper air-passages, and can often be relieved; but cough associated with signs of persistent diffuse bronchitis is often indicative of widely disseminated tuberculous lesions.

The state of the digestion is of the greatest importance. Where the symptoms of gastric disorder, or of faulty absorption or assimilation, prove rebellious to treatment the prospects of improvement are small indeed.

In attempting to weigh the indications of physical examination of the lungs, the two chief points requiring attention are the character and the extent of the disease. An acute onset is commonly followed by progressive invasion of both lungs, and has the gravest significance. An insidious, bronchitic, or hæmoptoic onset is more favourable. Rapidly extending disease is always of ominous significance. Râles and other morbid signs scattered widely over a large part of both lungs, especially in pyrexial cases, point to disseminated lesions, a most unfavourable type of disease; but similar physical signs, without much fever, may sometimes persist for months or years in cases where the disease takes the form of discrete fibro-caseous or fibroid processes. Cases with severe symptoms and relatively slight physical signs are to be regarded with suspicion, for the true extent of the pulmonary disease is generally masked by other conditions; on the other hand, the presence of marked signs of consolidation or excavation of one upper lobe is not inconsistent with a chronic and favourable course so long as the lower lobe and the opposite lung remain comparatively free.

Signs of contraction are a sure index of chronicity. Localised and stationary disease is a good element in prognosis. The disappearance of râles is, in general, a favourable feature.

After what has been said in a previous section about the stages of phthisis, it is futile to base the prognosis on considerations which are so apt to be fallacious. If, in a chronic case, we could be sure, which we cannot be, of the absence of softening and excavation, the prospects of arrest would be better than if cavities had already formed, for the existence of a cavity carries with it the risk of extension by means of inhalation of infective secretions into distant bronchi. Moreover, there is no evidence that a vomica can become obliterated by cicatrisation; whereas we know that tuberculous nodules often undergo healing by encapsulation, calcification, or fibrous transformation.

Among the most ominous complications are meningitis and pneumothorax. Pleurisy with effusion sometimes appears to exert a retarding influence on the pulmonary affection. Empyema is unfavourable. Dry pleurisy is regarded by some authors as a very unfavourable sign; but this is by no means generally true. The appearance of the diffuse infiltrating form of laryngeal tuberculosis, with its tendency to produce dysphagia and stenosis, betokens a speedy termination. Oft repeated hæmoptysis depresses the patient morally as well as physically; and under such circumstances the possibility of a sudden and fatal issue has always to be reckoned with.

Tuberculous peritonitis and intestinal ulceration cause great wasting and prostration, and generally hasten the patient's end. Tuberculosis of the abdominal lymphatic glands and generative organs tends to aggravate the general condition, and is commonly a sign of generalised disease.

Pronounced lardaceous disease of the viscera is a most serious complication of chronic cases, pointing, as it does, to profound derangement of nutrition. A combination of diabetes and phthisis is also a most grave condition.

The presence of cardiac hypertrophy and dilatation, or of marked emphysema, justifies the opinion that the duration of the disease will be long.

The environment is a matter of much importance. A patient living in a healthy country place, under suitable climatic conditions, has better prospects than one who is compelled to dwell in a large town, especially if his life be spent in dusty or smoky rooms. Again, pecuniary means have a direct bearing on the prognosis: those who can procure, not only the necessaries, but also the luxuries of life, and can afford to rest, are in a better position to battle with the disease than those who must work hard for a living. Nevertheless, among poor hospital patients we see, not very infrequently, persons who have been suffering from phthisis for ten years or more, and who still go on working under the most adverse circumstances. A history of previous good health is a hopeful feature, as a greater capacity of resistance may be expected where the general health has not been already undermined. The influence of age has been much disputed. As a general rule, pulmonary tuberculosis runs a more rapid course in children and young adults than in older persons, among whom the chronic form is rather the rule. Cornil and Hérard suggest that tuberculosis is more chronic in old people, because heredity has already weeded out those of least resistance. Nevertheless, acute disease may occur in elderly patients, and, conversely, the phthisis of children may be chronic. Each case must be estimated on all the data; and the influence of age can only be credited with a very subordinate importance.

It has been said that the duration of the disease is shorter in women than men. If we exclude the cases associated with pregnancy and parturition, it is doubtful whether this statement be true.

The influence of heredity is undoubtedly an important one. It is a common belief that this factor determines the earlier manifestation of the disease. A strong predisposition is an unfavourable element, as in such cases there often appears to be a general lack of vitality and resistance. But, although this is generally true, hereditary influence cannot be ranked on a level with considerations derived from a careful estimation of the effects of the disease in the individual patient. The best results may be expected in cases presenting the following features:

—apyrexia, or a subfebrile temperature; weight stationary or increasing; signs of disease confined to one lung or to limited portions of both lungs (especially if associated with contraction); a quiet pulse and nervous system; a good digestion; absence of serious complications; a good family and personal history, and favourable hygienic surroundings.

Treatment.—A. *Preventive.*—If, as our present knowledge appears to show, the sputum of tuberculous persons be the main source of the disease, it is obvious that the complete destruction or disinfection of this secretion should be our first duty. In many hospitals this is effected by means of special destructors, or furnaces, in which the sputum is burnt. In private houses, where this method is difficult of application, the expectoration, after previous disinfection, may be discharged into the drains. For general purposes carbolic acid in a strength of 5 per cent is the best disinfectant; experiment shows that the infectiveness of the bacilli is completely removed after exposure of the sputum to this solution for a short time. Sputum should not be thrown on the dust-bin, where the contents may dry and become a further source of danger. In all cases, whether in hospitals or private houses, patients should be directed to use spittoons containing a suitable disinfectant. If, in spite of advice to the contrary, patients use handkerchiefs for receiving the sputum, these should be burnt; or at any rate should be scalded before being sent to the wash.

Persons suffering from phthisis should be warned not to spit about the streets, or the house, or into any vessel which does not contain some disinfectant. Underclothing, linen, sheets, and pillow-cases should also be scalded before being washed, especially in the case of bed-ridden patients, when the chances of contamination are greater. Phthisical persons should occupy separate beds. Bedrooms and sitting-rooms so occupied must be carefully cleaned with a damp cloth, so as to avoid raising a dust; and should be well aired and exposed to light every day. Rooms that have been inhabited by such patients should be thoroughly cleaned, and, if possible, white-washed, painted, and re-papered before being used by other persons.

It is desirable that patients should be provided with separate sets of knives, and forks, and spoons; but, in default of this precaution, all table utensils, as well as plates, cups, glasses, should be scrupulously cleaned.

Milk is undoubtedly a vehicle of disease, and should be carefully boiled; particularly when intended for children. For the principles on which slaughter-houses and dairies should be regulated, and for further information on the general question of prophylaxis, the article "Tuberculosis" (vol. ii. p. 30) should be consulted.

In persons threatened with tuberculosis, and in others with a strong family predisposition, the importance of a good general hygiene can hardly be over-estimated.

Abundance of fresh air in the dwelling—especially in bedrooms—secured by suitable methods of ventilation, a large amount of outdoor life in pure country air, a generous diet, including a large proportion of fatty constituents, daily cold sponging of the body, and the use of flannel or similar underclothing, are amongst the most necessary conditions. In the case of children the throat needs special attention; enlarged tonsils should be removed, and catarrhal affections must not be neglected. The opinion is gaining ground that the tonsils are frequently the portals by which tubercle bacilli enter the body; at any rate, in primary tuberculosis of the cervical lymph glands. The question of the removal of caseous glands, and the surgical treatment of tuberculous disease of bones and joints, are matters of great importance, but cannot be discussed here. [*Vide* vol. iv. p. 599.] Tuberculous mothers ought not to suckle their infants.

The choice of a profession or trade is a matter of no small consequence. Occupations in which life is mainly or largely spent in the open air are the most favourable; but, in the case of the poor, outdoor work generally implies more or less heavy labour, which is often prohibitive under the circumstances. Many people, in whom tuberculous affections of bones, joints, or lymphatic glands have been cured or partially arrested, manage to carry on successfully various sedentary trades or professions. Dusty occupations, as in the case of millers, bakers, knife-grinders, stone-masons, and the like, are fraught with special dangers to vulnerable persons. Free ventilation of dusty workshops is all-important, and serves to minimise, to a large extent, the dangers of the aforesaid trades.

There can be no doubt whatever that persons suffering from progressive disease ought not to marry. In cases of quiescent or apparently arrested tuberculosis there is room for difference of opinion. When all symptoms of disease have disappeared, the sputum no longer contains bacilli, and the general health remains good, marriage, in the case of men, may be undertaken after the lapse of two or three years without any great risk. Women incur far greater danger in connection with pregnancy, parturition, and lactation; for it is well known that, under the influence of such conditions, quiescent tuberculous lesions are apt to prove the starting-point of active disease. If, however, the tuberculous process can only be regarded as quiescent, and bacilli continue to be expectorated, marriage ought to be forbidden in either sex. Most writers agree on this point as regards women; but some have urged that men, under these circumstances, may be allowed to marry on the ground that their lives are thereby made happier; and that, if children should be begotten, they tend to die off early, and the race does not appreciably suffer. The morality of such advice need not be discussed here; but the possibility of a phthisical husband directly or indirectly infecting a healthy wife cannot be disregarded; and the risk of adding to the already high tuberculous death-rate is one that no medical man should willingly countenance. However, as all writers point out, the question of marriage is seldom decided, solely or even mainly, on medical grounds.

B. *Specific treatment.*—It has been well said that where the number of remedies recommended for any disease is large, there is good reason for the belief that none of them is possessed of much efficacy. Of no disease may this more truly be said than of tuberculosis. Tuberculin, a glycerine extract of pure cultures of tubercle bacilli, deprived of the bacilli by a special method of infiltration, and injected under the skin, was found by Koch to exert a marked influence on tuberculous lesions. The local action of the remedy consists in an inflammatory swelling and disintegration of the diseased foci. Constitutional reaction is indicated by fever, malaise, headache, pains in the limbs and trunk; and occasionally by nausea and vomiting. The effects of the remedy were most conspicuous in cases of lupus, where the changes in the skin could be readily followed.

Although Koch's statement that tuberculin had a specific influence on tuberculous lesions was speedily verified, his claim that the action was curative was not so generally admitted. Pathological evidence was soon brought forward by Virchow and others to show that the use of tuberculin was often followed by the development of acute inflammatory changes in the lungs; and that under its influence quiescent disease may spring into activity and lead to generalised tuberculosis. Space will not admit of a detailed discussion of this matter. The prevailing opinion at the present time is that the administration of tuberculin in cases of pulmonary tuberculosis is dangerous, though some surgeons still regard it as a useful adjunct to other methods of treatment in surgical forms of tuberculosis.

Klebs and Dr. W. Hunter, working independently, claimed to have succeeded in separating the fever-producing and toxic elements from the direct curative constituents of tuberculin; but their extracts failed when put to the test by other observers. Quite recently Koch has described a new method by which he was able to prepare an improved tuberculin. Dried cultures of the tubercle bacillus were thoroughly triturated in a mortar, then mixed with distilled water and centrifugalised. The sediment was again dried, triturated, suspended in distilled water, and centrifugalised. This procedure was repeated until no sediment remained. The liquid separated by the first centrifugalisation contains the active principles of the original tuberculin, while the fluid obtained at subsequent stages of the process is believed to contain the débris of the disintegrated bacilli themselves. This solution has the great advantage that it produces no constitutional disturbance beyond a slight rise of temperature. The new tuberculin was found to confer immunity on guinea-pigs, and its use in the human subject in cases of lupus and early pulmonary tuberculosis was followed by improvement. Further experience can alone decide as to the therapeutic value of the new preparation; but it is no exaggeration to say that in a further development and improvement of Koch's method lies our best hope of arriving at a successful treatment of tuberculous disease.

The number of drugs that have been vaunted as specifics for tuberculosis is legion. In recent years iodine, iodoform, carbolic acid, corrosive sublimate, creasote and one of its constituents, guaiacol, have been most largely used, in virtue of their antiseptic properties. These have been administered by the mouth, by inhalation, subcutaneous injection, inunction, and direct injection into the lung; and sulphuretted hydrogen gas has even been pumped into the rectum. The results have not differed greatly in respect of any of these methods. They have all passed through successive stages of exaggerated and hasty laudation, half-hearted approbation, and contemptuous neglect. The history of guaiacol is a good illustration: at first it was advocated as a specific; now the only claim seriously made in its favour is that it has a beneficial effect on cough and expectoration.

As we have no specific remedies, our aim must be to increase, as far as possible, the resisting power of the patient, so as to put him in the best condition to withstand the inroads of the disease.

In order to maintain the nutrition of the body at as high a level as possible, the dietary must be liberal, and should include a large amount of fat in the shape of milk, cream, butter, fat bacon, and the like, in addition to a due proportion of nitrogenous and carbohydrate constituents. A special distaste for fatty foods is manifested by some patients, but this aversion is by no means so general as certain writers would have us believe. Cod-liver oil is a valuable adjunct to the diet, but is possessed of no specific virtues. This valuable food is sometimes prescribed in a manner calculated to bring it into discredit, that is, when it is administered in too large quantities. Two to four drachms twice or thrice in the day is as much as most patients can digest, and it is frequently necessary to begin with even smaller doses. It usually agrees best when taken soon after a meal; but some people prefer a single dose at bedtime. In all cases of dyspepsia, and whenever the taste of the oil keeps rising into the mouth, it should be withheld. If persevered with under these circumstances it seldom fails to derange the digestion. It is better for the patient to enjoy his food without the oil, than to persist in its use and lose his appetite.

The taste of the oil may be disguised with peppermint, lemon juice, ginger or orange wine, cognac, liqueurs, and other flavourings. Many patients prefer to take the oil in the form of an emulsion, or in combination with malt extract. Malt is much used at present in England, and no doubt it possesses some digestive value; but it is no substitute for cod-liver oil or fats. Glycerine, in doses of three to four tablespoonfuls daily, has been recommended as a substitute for the oil by Jaccoud, but it has not found much favour with other physicians. Alcohol is not to be ordered in all cases indiscriminately; where the disease is quiescent, nutrition fairly well preserved, and the appetite good, it is not required; in conditions of debility, deficient appetite, and, above all, in pyrexial cases, alcohol is of great value.

The particular form in which stimulants are to be administered is largely a matter of taste. Ale and stout are preferred by many patients. In pyrexial cases brandy, whisky, or some form of spirit, seems often to suit best; and the quantity that can be taken with advantage under these circumstances may be very large. Alcohol has no influence in promoting reparative sclerotic changes, as some have asserted. In cases of obstinate anorexia forced feeding by means of the stomach-tube has been found useful by Debove and others. Massage is occasionally useful, especially where debility is a prominent symptom, and is unaccompanied by pyrexia.

In addition to the utmost attention to the matter of food the rules of general hygiene must be carefully observed. The patient's house ought to be well drained, built on a light porous soil, and, if possible, it should face the south. The rooms, and especially the sleeping-apartments, must be well ventilated and suitably warmed. It is hardly possible to over-estimate the value of fresh air and sunshine. Regular exercise, walking, riding, outdoor games of the less violent kind, such as golf, cycling, shooting, and fishing, may all be practised with moderation if the patient's general condition be good, and if there be no pyrexia. Where this is not possible, the patient, in favourable weather, may go out in a bath-chair or in a carriage; or he may sit out of doors in a suitably arranged shelter. Even when he is entirely confined to bed with fever, wasting, night sweats, and symptoms of progressive disease, much benefit may still accrue from wheeling the patient's bed out of doors into a sheltered spot, or into a sunny balcony, as is done at Falkenstein and other places; bed-ridden persons may, in this manner, spend the greater part of the day in the open air with great advantage. The clothing should be warm and yet light, and woollen garments should be worn next the skin. Comforters for the neck and chest-protectors, which encourage hyperæmia and increased sensitiveness of the skin to changes of temperature, are unnecessary and unadvisable. Woollen socks or stockings and thick boots are required to prevent the feet from getting chilled. Respirators worn over the mouth are not now so much in vogue as formerly. If the patient breathe through his nose, as he ought to do, a respirator is superfluous. When a strong, cold wind has to be faced, a light shawl or thick veil may be wrapped round the face for temporary protection.

The skin may be rendered less sensitive to changes of temperature by the daily use of the cold shower bath or douche in the morning; but in the case of more delicate patients, with feeble circulation, a warm bath followed by cold sponging is preferable. Early hours, the avoidance of crowded rooms, theatres, and smoking-rooms, a life free from excitement, and occupation for the mind, such as reading, drawing, chess, billiards, and other indoor games, are to be recommended. Instrumental music may be practised, but singing is not advisable, except for the more robust patients, in whom the disease is quiescent; though Walshe gives instances of singers continuing to take leading parts in the opera while suffering from pronounced pulmonary disease. Medical direction is most desirable; and the success of some Continental health resorts is doubtless attributable in a large measure to the careful and strict superintendence of the physicians in charge. But the majority of English patients find such a rigid supervision irksome and disagreeable; and hitherto such establishments have not been in much request in this country.

In selecting a suitable climate we must be guided by certain general principles. Purity of the atmosphere, and especially freedom from dust of all kinds, and abundant sunshine, are the fundamental requisites. Questions of altitude, temperature, and moisture of the air, and geographical considerations in general, are still matters of dispute, and are discussed elsewhere in this work.

If the patient's surroundings satisfy the requirements just indicated, it is unnecessary, in many instances, to advise a permanent change of residence. In the case of wealthy people it may be desirable to send them away to some health resort, where they will be more ready to submit to strict medical supervision and direction than at home. When the patient lives in a large town he should be recommended to remove into purer air, if his means permit. It is useless and cruel to send patients with advanced disease to a distant health resort. To such persons the fatigue of a long journey may have disastrous consequences, and the loss of home comforts cannot be compensated even by the best of climates.

If the general health be well maintained, and the pulmonary disease be neither very active nor extensive, great benefit may be obtained by spending the winter, or better still, by continued residence at the high alpine stations, such as St. Moritz, Davos, and the like. Better results are generally obtained in men than in women, as the tastes and the habits of men impel them to take a fuller advantage of the opportunities of outdoor life and exercise presented by an alpine climate. Under similar conditions of health, emigration to Colorado, the Rocky Mountains, and the high levels of South Africa offers good prospects to young men.

Emphysema, laryngeal tuberculosis, and manifestations of nervous erethism are generally regarded as contra-indicating residence at the high alpine stations. Such cases are more adapted for Egypt, the Riviera, Madeira, the Canaries, or the south coast of England. For a detailed discussion of this subject the reader is referred to the article, "Climate in the Treatment of Disease," vol. i. p. 247.

C. *Symptomatic treatment.*—In combination with the general hygienic measures that have been briefly sketched the exhibition of certain tonic drugs is often very useful. The most valuable are strychnine or nux vomica, arsenic, and quinine. Opinions differ as to the relative value of these, but strychnine appears to deserve the first place. Arsenic has no specific influence on the disease, but it may do good service in its capacity of a nervine tonic; the same may be said of quinine. Iron has still a great reputation with some physicians; but it does not suit all patients, especially the large class that suffer from a tendency to gastric catarrh; and it has little effect on the anæmia of phthisis. In persons who can take a fair amount of exercise, and have a good digestion, a short course of iron, either alone or in combination with arsenic, is sometimes attended with good results. The hypophosphites of lime and soda have been largely tried, and are still much used in this country. They are certainly not possessed of any direct action on the tuberculous process, and their tonic effects have been greatly over-estimated.

Fever.—Quinine has been extensively used for the purpose of reducing fever, especially on the Continent; but it is generally allowed that in order to obtain this result 20 to 30 grains must be given in a single dose, or divided into four or five doses, to be taken at short intervals some hours before the temperature begins to rise. Even when administered in such quantities the antipyretic action of quinine is but slight, and the stomach is often deranged by the drug. The combination of quinine, opium, and digitalis, known as Niemeyer's Pill, has long enjoyed a great reputation in pyrexial cases; but, although its general effects are sometimes salutary, it is rarely very efficient in the reduction of temperature.

Of late no small number of antipyretic drugs have been employed—salicylic acid, salicylate of soda, antipyrin, thallin, phenacetin, antifebrine, and many others. The most effectual seem to be antipyrin and antifebrine, which, when given in sufficient doses, undoubtedly effect a considerable fall of temperature. Antifebrine is a very powerful remedy, but its action is somewhat uncertain. It is never advisable to prescribe larger doses than two or three grains to begin with: in these quantities it may be repeated at short intervals till eight or ten grains have been taken. Unfortunately the reduction of temperature produced is but temporary, and no further effect on the disease is produced. Moreover, the prolonged use of antifebrine and antipyrin is extremely depressing, and causes profuse sweating. Wilson Fox believed that the continued use of small doses of these remedies and of salicylate of soda had a beneficial result on the general condition, although the range of temperature was not appreciably affected.

Tepid sponging during the pyrexial periods sometimes gives considerable relief, even if it fail to reduce the temperature of the body to any great extent. The use of the cold or tepid bath finds few advocates on account of the further depression which, in the prostrate condition of such patients, is apt to follow its use.

Sweats.—Atropine in doses of $\frac{1}{100}$th to $\frac{1}{60}$th of a grain, given at bedtime, is the most effectual agent we possess for checking sweats. A combination of extract of belladonna and oxide of zinc is also useful, but it is inferior to atropine. Picrotoxin, $\frac{1}{100}$th to $\frac{1}{60}$th of a grain (Murrell); strychnine, 10 ♏ of the liquor (Lauder Brunton); and agaricin may also be employed with advantage in some cases. In the slighter cases, to sponge the skin with toilet vinegar and water may be sufficient. Arsenic is recommended by some authors, but when the sweats are profuse it has little influence.

Cough.—A moderate cough is the natural consequence of pulmonary disease, and needs no special treatment; moreover, where secreting cavities exist, effective cough is most desirable. But when the cough is very violent, spasmodic, or incessant, and the patient becomes much exhausted thereby, it is necessary to treat this distressing symptom directly. In order to do this with success we must first discover the source of the cough. The more violent the fits of coughing, the more likely are we to find that the cause is situated in the larynx or main air-passages. When the larynx is the seat of ulceration or inflammation local treatment is indicated. Among the most useful sedative remedies we may mention an intra-laryngeal spray of cocaine (2 per cent solution), an inhalation of 10 drops of oil of peppermint, or of a 20 per cent alcoholic solution of menthol in an orinasal respirator, and the use of 1 drachm of glycerine of carbolic acid, with 10 drops of chloroform added to half a pint of boiling water in a steam-inhaler. Creasote or carbolic acid, diluted with rectified spirits, or spirits of chloroform, may also be used for inhalation in an orinasal respirator. In similar affections of the trachea and large bronchial tubes the same treatment may be applied; but the cocaine spray can only reach the upper part of the trachea at farthest.

Where the cough depends on bronchitis of the smaller tubes the treatment is that of ordinary bronchitis; an alkaline mixture containing bicarbonate of soda, or citrate of potash, with a few minims of ipecacuanha wine, may be prescribed; and, if expectoration be difficult, 3 or 4 grains of carbonate of ammonia may be added. In some cases 5 ♏ of antimonial wine may be substituted for ipecacuanha for a few days with excellent effect. Similar drugs may be prescribed in an effervescing mixture. Iodide of potassium in small doses, squill, and senega may also be given when secretion is tough and difficult of removal. Warm drinks, like tea, cocoa, or milk, or a steam-kettle to moisten the air, may often be used successfully for the same purpose, and may be tried before resorting to expectorant remedies. But in many cases all the above-named measures fail to give more than temporary relief, and sedative drugs are required. A linctus containing tincture of belladonna, spirits of chloroform, and glycerine may sometimes prove useful; but in the worst cases opium in some shape is indispensable. A combination of morphia and hydrocyanic acid with glycerine, spirits of chloroform, or syrup of wild cherry, forms an effective linctus, which, however, must not be used too freely lest the digestion be deranged.

Codeia may be substituted for opium, as it interferes less with the appetite; but its sedative effects are not equal to those of morphia.

In some cases of early disease, associated with a troublesome cough, much relief may be obtained from a small blister applied to the subclavicular region on the affected side.

Expectoration.—When the expectoration is very profuse, the administration of purified creasote or guaiacol in capsules is sometimes effectual in diminishing the excessive secretion of the bronchi and pulmonary cavities. Turpentine and various resinous drugs are sometimes given for the same purpose; but the expectoration is symptomatic of broncho-pulmonary disease, and rarely requires direct treatment.

Dyspnœa, though seldom a prominent feature, is occasionally very distressing. When due to acute miliary tuberculosis and rapidly advancing pulmonary disease it admits of little relief, and we must be content to administer stimulants; such as ammonia, ether, and brandy.

Attacks of dyspnœa, depending on violent and ineffectual attempts to remove tenacious secretion from the bronchial tubes, may be mitigated by the judicious use of expectorants, the best being ammonia and senega. Steam inhalations of carbolic acid may render good service. In some cases dyspnœa has been lessened by inhalations of iodide of ethyl, as suggested by Dr. R. E. Thompson. Dyspnœa arising from cardiac failure, with attendant œdema of the lung, must be treated by diffusible stimulants and hypodermic injections of strychnine. The dyspnœa of pneumothorax will be referred to presently.

Hæmoptysis.—The pulmonary hæmoptysis of tuberculous disease may be due to capillary hæmorrhage, ulceration of vessels, or aneurysm of the pulmonary artery. The loss of blood in the first case is never extensive, and direct treatment is not required. In the second and third cases hæmorrhage is the result of gross lesions of comparatively large vessels, consequently the amount of blood lost may be considerable; yet even under such circumstances spontaneous cure is not infrequently effected by the formation of a thrombus, which seals up the ruptured vessel.

In our treatment of hæmoptysis we endeavour to imitate nature's method, that is, to promote thrombosis by lowering the pressure in the pulmonary artery.

It is important to recognise that the faintness which often attends the attack is a symptom of cardiac depression—a condition in itself favourable to thrombosis. In all cases, whether the hæmorrhage be profuse or slight, absolute rest must be insisted upon. The patient must keep in bed in a cool airy room, and should maintain a semi-recumbent position. Talking, movement, or excitement of any kind must be avoided. Nothing more than iced milk, meat jelly, and small sandwiches of bread and butter should be given for the first few days, and alcohol in any form must be expressly forbidden. The cough, which is rarely absent, may be relieved by sucking ice; but when it cannot thus be checked some preparation of opium must be administered. Small and frequent doses of morphia may be given by the mouth, or one-third of a grain may be injected under the skin. This remedy not only exerts a valuable local effect on the injured vessel by the rest which it gives to the lung, but it helps also to allay the restlessness and agitation of the patient. At the same time, seeing that in most fatal cases of hæmoptysis death occurs from suffocation rather than from the amount of blood lost, it is clear that the indiscriminate use of morphia is not without its dangers.

When from flooding of the bronchial tubes with blood dyspnœa is very pronounced, cough should not be checked by sedative drugs. With the view of reducing the blood-pressure, sulphate of soda or magnesia, in doses of 60 grains, should be given every three or four hours to begin with. Saline purgatives cause determination of blood to the intestine, and thus relieve vascular tension, but, unfortunately, their action is rather slow. Aconite has been recommended by Dr. Andrew, on the strength of experiments by Dr. Bradford and Mr. Dean which show that it causes a fall of pressure both in the carotid and pulmonary arteries. This method seems worthy of trial, but I have not had sufficient experience of it to express an opinion as to its merits. Astringents, like gallic acid and lead, are still extensively used, but it is difficult to see what effect they can have on aneurysms or ulcerated vessels. Ergot is perhaps the most popular drug at present, but, according to Bradford and Dean, it causes a rise of blood-pressure not only in the aortic but also in the pulmonary circuit; a result which must aggravate rather than check hæmorrhage from the lungs. Clinical experience shows that the effect of ergot is as uncertain and unsatisfactory as those of gallic acid and lead.

The constipation which is produced by the last two remedies must tend, moreover, to raise blood-pressure, which is injurious. Oil of turpentine in large doses sometimes does good service, probably in virtue of the cardiac depression which it causes. Nauseating doses of ipecacuanha, recommended by Trousseau, seem to act in the same way; but the risk of vomiting is a serious one, and the remedy is now seldom employed. The application of ice to the chest appears to be of very doubtful utility, and in this country is little used.

The artificial induction of pneumothorax to cause collapse of the lung and pressure on the ruptured vessel was unsuccessfully employed by Dr. Cayley in one case of persistent hæmoptysis. Under similar circumstances it would be worth while, in conjunction with other measures, to try Prof. A. E. Wright's plan of administering chloride of calcium in 15-grain doses three times a day, for a few days, to increase the coagulability of the blood. In any case great care is needed in the management of the patient after the hæmorrhage has ceased. The diet should be very sparing, and the patient should not rise from bed for three or four days at least. Free action of the bowels should be secured by the continued use of saline laxatives. Alcohol should be avoided altogether for some weeks.

Pleurisy.—For the relief of the pain of dry pleurisy Dr. F. T. Roberts's plan of strapping the affected side gives excellent results, and can be strongly recommended. But in cases where one lung is extensively diseased, and pleurisy attacks the opposite side, it may not be possible to apply strapping without dangerously curtailing the already restricted respiratory surface. Under such circumstances we must be content with counter-irritation, a few leeches, and poultices; if these fail, a hypodermic injection of morphia will be required.

Roberts's method is specially adapted for the treatment of pleurisy affecting the lower part of the chest, where the ribs are more yielding and their movement easily restrained. Pleurisy in the region of the upper three or four interspaces seldom causes such acute pain, as the range of movement of the upper ribs is limited; here counter-irritation is generally sufficient. In cases of sero-fibrinous or hæmorrhagic effusion paracentesis should not be resorted to unless the quantity of fluid be so great as to cause embarrassment of the respiration. Experience shows that the pressure resulting from pleuritic effusion promotes arrest of the tuberculous process in the corresponding lung. As a rule the fluid is slowly absorbed.

In the comparatively few instances of advanced phthisis in which empyema occurs, the pleura should not be opened unless the abscess point externally, or unless the effusion be so large as to constitute a mechanical hindrance to respiration. In the latter case aspiration is preferable to free incision. The empyema once opened will rarely close again; and free incision appears rather to hasten the patient's end. In cases of early or limited pulmonary disease empyema must be treated on ordinary lines. It occasionally happens that the pus spontaneously becomes inspissated, and undergoes a caseous change.

Pneumothorax.—When pneumothorax arises acutely, with severe dyspnœa and symptoms of shock, stimulants in the form of brandy, ether, or ammonia should be given at once. Morphia has been recommended by some writers to minimise the effects of shock, but in the presence of marked dyspnœa an opiate is contra-indicated. Where the opening is valvular, and air accumulates in the pleural cavity under great pressure, paracentesis may be necessary in order to withdraw a sufficient quantity of air to relieve the pleural tension. Sir R. Douglas Powell advises that the side be afterwards strapped to prevent reaccumulation of air. Paracentesis is sometimes followed by subcutaneous emphysema. In the event of sero-fibrinous effusion, tapping may be successfully employed, but seeing that spontaneous recovery may ensue, it is well to wait for a time before resorting to this measure. In pyopneumothorax incision is generally considered to be unadvisable, and this, no doubt, is true of advanced cases. But the practice of early drainage is worthy of further trial, where the general condition of the patient is fairly satisfactory and the lung is presumably not much affected.

Laryngeal tuberculosis.—The treatment of this complication may be general and local. The general treatment is practically that of pulmonary tuberculosis, with certain reservations as to climate. The best atmospheric conditions are a temperate climate, a moderate degree of moisture in the air, and an absence of dust. At the same time, it may be admitted that many cases do well in such a dust-laden and apparently undesirable atmosphere as that of London and other large towns. Tobacco-smoking requires a brief notice. Many patients have no desire to smoke, but some have a craving for tobacco: if so, the patient may be allowed to smoke once or twice a day after meals, provided it be out of doors or in a large, well-ventilated room; the object of these restrictions being to prevent the inhalation of smoky air into the larynx and lungs. The practice of inhaling tobacco smoke should be forbidden. Strong alcoholic drinks, spices, and highly seasoned dishes irritate the pharynx and epiglottis, and are to be avoided. The local treatment is fully described in the chapter on "Tubercle of the Larynx," vol. iv. p. 800.

I may say here that to palliate the laryngeal irritation we may prescribe steam inhalations containing carbolic acid and chloroform; or at other times menthol or oil of peppermint on a respirator.

To soothe the pain so often present a 2 per cent spray of cocaine may be used a few minutes before meals. The local application of menthol, in the form of a 10 to 20 per cent solution in olive oil, sometimes gives relief; or, again, the insufflation of one-sixth of a grain of morphia with a little starch powder or sugar of milk. Some patients find benefit from sucking ice. When, in spite of these measures, the patient is unable to swallow, Dr. Wolfenden's plan may prove successful; the patient is directed to lie on his face, with his head over the edge of the bed, and to drink through an india-rubber tube. It may ultimately be necessary to have recourse to nasal feeding, which, at times, does excellent service. The operation of tracheotomy is very rarely required, the only indication being afforded by the existence of severe laryngeal stenosis and impending asphyxia. The treatment of tracheal tuberculosis can only be palliative.

Tuberculous ulceration of the pharynx, palate, and tongue must be dealt with in the same way, and with the same reservations as in the case of the larynx. Good results sometimes follow the use of lactic acid when the ulceration is localised and the subjacent infiltration is not very great. Granular pharyngitis and other non-tuberculous affections of the pharynx, which may give rise to troublesome cough and other symptoms, must be treated on the principles laid down in the article "Pharynx" (vol. iv. p. 729).

Gastro-intestinal symptoms.—Loss of appetite, cardialgia, and other symptoms of dyspepsia may be treated by alkaline and acid tonics; but for general use nothing can excel an alkaline mixture consisting of bicarbonate of soda (15 grains), tincture of nux vomica (10 minims), and compound infusion of gentian (1 ounce), given before meals. If a sedative action be desired, dilute hydrocyanic acid may be substituted for nux vomica. The good effects of this mixture are witnessed not only by increase of appetite and relief of the dyspeptic symptoms, but at the same time expectoration is facilitated, whereby the cough is indirectly relieved.

In other cases, especially where flatulence is a prominent symptom, better results are obtained by acids, with or without strychnine, given after meals. In cases marked by irritative symptoms—such as vomiting or pronounced epigastric pain and anorexia—a mixture containing bismuth, hydrocyanic acid, tincture of belladonna, or, if necessary, a few minims of liquor morphinæ, given before meals, is to be preferred. Bismuth in powder, or in an effervescing draught with hydrocyanic acid, may prove more successful in particular cases.

In all instances of dyspepsia the diet requires a careful survey. The diet should be light and digestible, and the meals small and more frequent than in health. In the comparatively uncommon form, distinguishable by a red glazed tongue, vomiting and anorexia, liquid food, especially milk with lime-water or soda-water, koumiss, veal or chicken broth, will be required; and complete rest in bed should be enjoined.

Inasmuch as this kind of gastric disorder mostly affects patients suffering from pyrexia and other symptoms of progressive disease, the outlook is very grave unless the gastritis can be speedily removed so far as to enable the patient to digest an adequate supply of food. If the symptoms resist the measures indicated, it may be necessary to rely exclusively on peptonised nutrient enemas for a few days, giving only a little iced water by the mouth, for the relief of thirst. Fortunately this form of gastric disturbance is not of very frequent occurrence.

In most cases of obstinate dyspepsia mild purgatives are called for, such as a small dose of calomel (half a grain to a grain at bed-time), followed by a teaspoonful of Carlsbad salts, dissolved in half a pint of warm water, in the early morning. Violent purgatives should be carefully avoided altogether, owing to the risk of setting up intractable diarrhœa. A tumblerful of hot water, sipped at bedtime for a few nights, often gives great relief by washing out the stomach and removing remnants of undigested food which are apt to undergo decomposition, and thus to aggravate the catarrhal condition of the stomach. Dilatation of the stomach occurs occasionally, and washing out may be required; though the cases in which this operation can be recommended are very few, as the disturbance caused by the passage of the stomach-tube in feeble patients may be attended with serious consequences. Gastric digestion may be assisted by the administration of pepsin or papain, but, except as a temporary expedient, little benefit is to be expected from this line of treatment.

Diarrhœa is a symptom that should never be neglected; it should be treated by rest in bed and the application of warmth to the abdomen and extremities. In many instances it depends on slight errors of diet; and in such cases regulation of the diet, and a mild purge to free the intestine from irritating substances, may be all that is required in the way of treatment. For this purpose we may prescribe 2 drachms to half an ounce of castor oil with 10 minims of laudanum, or a small dose of calomel. If the diarrhœa do not speedily yield, bismuth should be given, in 20-grain doses, with a few minims of laudanum. In the far more serious case where diarrhœa is the result of tuberculous ulceration or lardaceous disease, powerful astringents, combined with opium, are indispensable. A mixture containing aromatic sulphuric acid, tincture of opium, and decoction of logwood, or again of subnitrate of bismuth, tincture of catechu, and tincture of opium, will often suffice to keep the diarrhœa in check. But in the most severe cases we must have recourse to stronger remedies, the best, perhaps, being a pill containing sulphate of copper (¼ grain) and opium (½ grain), given once, twice, or three times a day, as may be required. In cases where the ulceration affects the lower end of the colon the enema opii (B.P.) gives more relief than anything else. It will generally be necessary to revise the diet carefully; the most suitable food in the acute cases being milk, koumiss, or carefully prepared beef-tea; but when the diarrhœa lasts for weeks, boiled fish and tender meat, freed from fat and pounded or finely minced, may be given in small quantities. Digestion may be aided by peptonisation of the milk, and by the use of pepsine or papain after meals. The slightly bitter taste of peptonised milk may be masked by the addition of a teaspoonful of rum, cognac, or liqueur.

If the diarrhœa be accompanied by much pain, hot fomentations should be applied to the abdomen, and the warmth of the extremities sedulously maintained. In the rare event of serious intestinal hæmorrhage, an enema, consisting of a teaspoonful of oil of turpentine suspended in two ounces of starch mucilage, should be administered at once. Acute peritonitis must be treated on general principles by opium and hot fomentations. In the yet rarer instances in which perforation can be diagnosed the propriety of surgical interference must be considered; but the patient's general condition and the extent of the pulmonary disease may not justify such measures.

In chronic tuberculous peritonitis with effusion, whether serous or purulent, incision has, in several instances, been followed by arrest or cure. A similar result may also ensue without any surgical measures; in these cases, no doubt, the effusion is serous. Some physicians believe that the application of mercurial ointment to the abdominal wall promotes absorption of the fluid.

When suppuration has occurred, incision should not be delayed. Drugs seem to be of little use in this affection.

Renal symptoms. Albuminuria, whether due to nephritis or amyloid disease, is mostly found in advanced chronic cases. In such circumstances active treatment, by rigorous milk diet and purgation, is quite out of place. If possible, milk should be taken freely; but it is not advisable to prohibit a certain amount of meat and fish if the patient can take them. The drug treatment may include digitalis and iron, mild saline diuretics, and an occasional small dose of blue pill. But treatment should be addressed primarily to the general condition rather than to the renal disease, which is a local consequence of the constitutional malady.

In the few instances where nephritis occurs at an early period of the pulmonary disease, and where the health is not seriously affected, treatment may be conducted on ordinary principles.

The tuberculous pyelitis and cystitis of advanced cases do not admit of more than palliative measures. If the lung disease be slight, surgical advice should be sought.

Nervous symptoms.—For the treatment of meningitis, tuberculous tumours of the brain, and nervous complications in general, reference must be made to the proper articles.

It seems desirable, in conclusion, to sum up the general plan of treatment suitable for an ordinary case. In the first place, attention must be carefully directed to the rules of general hygiene; to the importance of spending as much time as possible in the open air, and the necessity of an abundant supply of food. Excepting in the most favourable instances, where the disease is quiescent, some form of tonic medicine will be required from time to time, the best being an alkaline bitter mixture, such as that already indicated. Narcotic and sedative drugs generally should be employed with great caution, because of their prejudicial influence on digestion; and complications, as they arise, must be treated on general principles.

PERCY KIDD.

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P. K.

PNEUMOCONIOSIS

PNEUMOCONIOSIS, pneumonoconiosis, or, translated into English, "Dusty-lung-disease," is a lesion that has attracted but little attention in this country—a circumstance the more remarkable considering that Great Britain has long held the first rank in manufacture, and that a large proportion of its population is consequently engaged in dusty occupations. At the same time, credit is due to British physicians for the early recognition of inhaled dust as a cause of lung lesion; among them may be mentioned Christison, Addison, Bennett, Corrigan, and Peacock, who taught that inhaled dust can penetrate the lung tissue, and that its presence can be demonstrated therein. This doctrine, however, had for a long time numerous adversaries, who argued that the black granules[1] so frequently met with were derived from the carbonaceous materials of the blood; and it is only within the last half-century that this opinion has been given up. That it held sway so long was owing to the influence of the eminent pathologist Virchow. Without denying that a black colour can be derived from the blood, it must be admitted, nevertheless, that the pigmentary particles generally found in the lungs, and especially in the lungs of persons engaged in dusty occupations, are derived from inhalation of dust.

The opposite opinion derived great support from the notion long held that the orinasal passages are so perfect and efficient as dust-strainers that, in co-operation with the cilia lining the bronchi, no dust could reach the deeper lung tissue. This opinion has been disproved both by experiment and by observation, and is no longer tenable. Further, although it is true that this conservative apparatus, so efficient in itself, is greatly re-enforced by the strong expiratory act of the lung, yet it will fail to arrest the ingress of particles if dust exist in large quantity or is breathed almost without intermission; or again, if the mucous membrane have suffered damage such as to facilitate its entrance into the submucous tissue. Under such circumstances the foreign matter enters the extravascular lymph-current and lymphatics, pursues its course along the pulmonary interspaces and connective tissue, and eventually reaches the alveoli and bronchial glands. The last-named organs act as barriers against its farther progress, and in consequence they become deeply coloured and swollen, and occasionally suffer ulterior changes.

Consolidation by the excessive growth of fibrous tissue is the chief pathological feature of pneumoconiosis. The pulmonary is transformed into fibrous tissue; the extent of change being dependent chiefly upon the physical character of the dust inhaled, but in some degree also upon accidental conditions of employment. The fibrotic change is almost always associated with thickened pleura, and the degree of this change bears some relation to the extent of fibrinous production in the lung substance itself. Now and then the fibrotic change seems to start from the pleura, and to spread in a branching, vein-like fashion; or in bands across and through the lung. The ramifying lines of fibrous growth for the most part represent interlobular or interstitial spaces, and are white in colour; this, however, is no essential character, for not infrequently the marble-like venation is black or brown, and its colour is largely dependent on that of the dust inhaled. The consolidation in question evidently has its origin in an exudation of lymph consequent upon very chronic inflammation of low intensity, due to the passage of dust into the bronchial tubes. A very similar consolidation, though rarely so extreme, follows the inhalation of tubercle bacilli; and as there is no little resemblance in many particulars between pneumoconiosis and tuberculous disease of the lung, there has been great confusion between the two—particularly on the part of laymen, who have not inaptly called both the one and the other by the common name of consumption. At the same time, as it was observed also that consumption, in this wide sense, manifests itself pre-eminently in certain occupations, further distinctions were made between that of potters, of stone-workers, and of Sheffield grinders;—"grinders' rot," "miners' rot," and so forth.

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[1] It is very gratifying to be able to refer to so admirable a collection of specimens of Pneumoconiosis as that in the museum of the Middlesex Hospital,—the result of long-continued pathological research made by the late Dr. Headlam Greenhow, physician to that hospital. Moreover, we have the benefit of his history of many of the cases that furnished those specimens recorded in the volumes of the Pathological Society of London. In no better way can the morbid anatomy of pneumoconiosis be studied than by an inspection of this collection; I have accordingly introduced the numbers affixed to some of the specimens in the proper places of this article.

The fibrosis in different cases varies greatly in extent, in density, and in the degree to which the bronchial tubes and pleura are implicated. The condensation in some instances does little more than destroy the sense of crepitation under the fingers; whilst in others the pulmonary tissue, losing its sponginess, is transformed into a dense mass which, in the most advanced specimens, shows no traces of normal structure, and in hardness, and often also in colour, resembles india-rubber. On section, moreover, this dense mass betrays at times an appreciable amount of grittiness, particularly if the dust inhaled be of a siliceous or metallic quality. The resemblance between specimens of pneumoconiosis and of tuberculous disease—especially where the latter has been very chronic—is often so great that, to the eye, the two may be almost indistinguishable, and the true nature of the disease a matter of doubt. In the eyes of many persons the two diseases are inseparable, and the opinion is held that the dust-made disease does not exist apart from tubercle. That pneumoconiosis, however, does exist apart from tubercle is the conclusion of a large number of independent observers. The determination of this problem in particular cases will depend, therefore, upon the discovery of the specific bacillus.

Sir Andrew Clark uses the name "phthisis" in a wide sense, and recognises two forms of it: (i.) the tuberculous, and (ii.) the fibroid, the former being characterised by the tubercle bacilli. Dr. Thomas Harris of Manchester, in his able lectures on phthisis, makes a parallel division of fibrotic pulmonary consolidation—under the appellation of interstitial pneumonia—into "primary" and "secondary." The former is represented by Corrigan's pneumonia, which makes its appearance without evident cause; the latter by pneumoconiosis, with an obvious cause in the inhalation of dust.

The grounds for the distinction made by both these authors are well set forth, by the former on clinical, by the latter on pathological data; both, however, agree that the non-tuberculous variety is of comparatively rare occurrence. As pneumoconiosis is primarily local, and without constitutional complications, all other viscera, except the lung affected, may remain normal and carry on their several functions, until indeed the long-persistent local derangement brings about secondary disturbance in one or more functionally connected organs. All such secondary disorders are of late appearance among the phenomena of pneumoconiosis; they may even extend to heart disease, with a certain amount of general dropsy, gastric and hepatic troubles, and secondary albuminuria. This being the case, the sufferer with pneumoconiosis is enabled to undergo considerable exertion for a long time, and is apt to look upon his earlier symptoms as no very important affair.

Although linked by its inflammatory characters to pneumonia, pneumoconiosis is not an example of croupous inflammation; it is rather a bilateral peribronchitis. Not infrequently it sets up a nervous disorder indicated by spasmodic breathing; hence it is also often called asthma, a name further distinguished by a noun indicative of the employment to which it is due. It is, moreover, a non-febrile malady; though there may be an intercurrence of active disease, the product of chill, or of supervening tuberculous deposit, with consequent elevation of temperature. In either case hectic symptoms may appear, local softening, and now and again a patch of ulcerative gangrenous decay. Pneumoconiosis is not a disease of children, but of adults, and these for obvious reasons are almost always of the male sex. The form of fibrosis which occurs in children after attacks of measles and whooping-cough is quite different from pneumoconiosis. Pleuritic thickening, as before said, is commonly met with; nevertheless it cannot be esteemed a necessary concomitant. The like is true also with regard to chest deformity. This last incident owns as contributory causes pleuritic adhesions and the shrinking of the lung itself as a result of progressive contraction of the fibrotic tissue diffused through its substance.

When once fibrosis has invaded the pulmonary substance, its tendency is to advance; chiefly because of repeatedly recurring bronchial attacks, due to fresh bronchial colds and the continued introduction of dust, intensifying the inflammatory action. For the most part both lungs become affected, though in varying degree. This would seem to be a necessary consequence of the entrance of the dust by the common channel of the larynx; nevertheless, some further determining cause operates to vary its diffusion, and to account for its predilection for the posterior and middle portions of the lungs. The chronic interstitial pneumonia of Clark and others is a unilateral disease generally due to a dry pleurisy.

Symptoms.—The disease is an extremely chronic one, and, beginning as a non-febrile bronchitis, it attracts little attention until an area of the breathing tissues of considerable extent is more or less disabled. The augmented bronchial secretion is at first noticed chiefly on waking, or on passing from a warm workshop into the open air. It is nothing more than ordinary mucus, with minute particles diffused throughout it, numerous enough in many instances, when the dust is of a dark hue, to give it a black colour; it is glairy, and is coughed up with some effort. As yet the affected workman does not suffer in his general health. He eats, drinks, and sleeps well, and joins in active physical exertion. But the conditions of employment, involving continuous exposure to dust inhalation, cause recurrent bronchial attacks, each in its turn damaging the lung more and more. Months and years may pass with but a slow increase of cough and spitting, though with an amount of dyspnœa exceeding that which is met with in ordinary bronchitis, and out of proportion to the severity of the evident organic changes. Constitutional symptoms are, however, still absent, and as appetite and digestion are good there is no wasting.

Continuance in the dusty occupation is soon attended by more and more copious expectoration, which gradually acquires a yellowish gray tint and the features of muco-pus. At length, however, the time arrives when impeded respiration, oft-repeated cough, loss of rest and appetite, and the discharge of muco-purulent fluid tell injuriously upon the general health and strength. The sufferer loses ground in all directions, he cannot pursue his work as heretofore, nor take outdoor exercise; he cannot even lie comfortably in the horizontal position. He seeks hot rooms and quietude, and becomes a valetudinarian, calling for medical treatment to relieve his cough and yet more to relieve his breathing. And now it is that loss of flesh and colour becomes apparent, whilst urgent and now constant dyspnœa confines him to his house or to his chamber. At this stage of the malady the name of consumption is applicable enough; though, as will presently be seen, sufficiently distinctive signs between the two maladies are discernible.

The great dyspnœa suggests the existence of pulmonary emphysema (1271), but the known pathology of fibrosis indicates that where emphysema exists, it is vicarious and comparatively insignificant. The dyspnœa is attributable to abridged respiratory area, to the choking of bronchial tubes by secretions, and, it may be, to cardiac mischief; and, as before stated, certain dusts, by the possession of special properties over and above the strictly mechanical, still further aggravate the difficulty of breathing, and assimilate it to the asthma of emphysema: an example of such properties is found in flax-dust. Moreover, the asthmatic state is not conjoined with the barrel-shaped thorax of emphysema: on the contrary, the lungs shrink as the diffused fibrin progressively contracts, and the bronchi, as the tissues are compressed and solidified, are distorted, dilated, and thickened (1274, 1279). The movements of the chest walls are crippled by the fibrous bands which pass between the costal and pleural surfaces. By other adhesions the lobes of the lungs also are distorted, drawn together, and compressed; and as a result their freedom of action and air capacity are seriously curtailed. All these conditions necessarily add to the difficulty of respiration (1274). If pneumoconiosis be unilateral, the fellow lung expands, and indeed may become truly emphysematous.

Pathology.—That a particle of dust, when it comes in contact with mucous membrane, will cause great irritation is a fact of everyday observation in the case of the conjunctiva. The irritation is immediate, and so severe as speedily to produce injection of the blood-vessels, and an outpour of tears from the lachrymal glands; if the disturbing cause be not soon removed, the phenomena of inflammation will set in with sero-purulent discharge, thickening of the conjunctival membranes, and effusion of lymph as a fibroid film upon the transparent cornea beneath.

All these phenomena fall within the range of unaided vision; and we are fully justified in concluding that something of the sort occurs when the mucous membrane of the respiratory passages is directly irritated, modified, as it may be in some details, by the special histological qualities of lung tissue.

The degree of irritation set up, and its consequences, will vary according to the physical, chemical, and physiological properties of the offending agent, the quantity introduced, the frequency of its introduction, and the period during which its action continues. For instance, there are dusts which are escharotic and damage at once the structures they fall upon. This is true of dusts both of mineral and organic nature. There are other dusts which damage by their chemical properties; and, again, others of animal origin which may inoculate or infect the system. Lastly, the dust of poisons may enter the body and display their respective effects, not only locally, but on the whole organism also.

The dust that has entered within lung tissue can be detected by the microscope; and may often be seen, under appropriate tests, to preserve both its physical and chemical properties. For instance, particles derived from coniferous wood have been identified by their gland-bearing fibres, and siliceous particles by their translucent appearance and their resistance to acids other than hydrofluoric. But the particles of dust are for the most part amorphous, and diffused sparsely in the tissue invaded by them (1272); or they are arranged in linear fashion, or collected in small masses, or in vein-like form marking out the lobules and alveoli within the interior of some of which they may be seen (1279).

The permeating dust is usually of a black colour, though where it possesses a distinctive hue of its own this peculiar tint pervades the altered tissue. (In specimen 1276 the lung is stained with carmine.) A blue black is frequently seen (1271), and a yellowish or buff hue is not uncommon; deposit of the latter colour is mostly seen in the lungs of quarrymen, or sometimes in accumulated matter suggestive of caseous transformation (1278). Yet even where the dust inhaled is itself of a pale colour, the parenchyma infested by it is more or less black or slate coloured; frequently this deep colour is not wholly derived from without, but owes its origin to some material collected within the living tissue; it is most likely a derivative from the blood.

The very clogged portions of black lung formerly met with in coal-getters were proved by Christison and others to be due to a great accumulation of coal-dust, the nature of which was demonstrable by combustion.

The lung of a young child, until about eight years old, has a clear pink colour; but about this age black pigmentary spots or lines appear in lobar and lobular spaces, mapping out the surface in irregular areas, and producing thereby a marbled appearance. As age advances, these dots and lines multiply and enlarge, and mostly in adult life produce a generally diffused dusky colour.

In persons engaged in a dusty occupation this coloration becomes progressively more pronounced; its tint varies according to that of the inhaled dusts, but a black colour largely preponderates. In the instance of workmen exposed to the dust of oxide of iron a reddish colour prevails.

The fine impalpable dust most people breathe is not the cause of the true pneumoconiosis we are concerned with. It blackens the lungs more or less, but these organs appear to be very tolerant of it; nevertheless, it may be that a lung deeply charged with black pigment is less efficient and less able to withstand inflammatory or other disease. When, however, a directly irritant dust is abundantly inhaled during some industrial process, an active morbid process is the result, one ending in structural lesions.

The source, form, and physical qualities of inhaled dust particles have furnished the basis for the classification of ensuing morbid consequences to lung tissue. Thus authors have described the results of inhaling siliceous particles as *chalicosis* or *silicosis*; of metallic particles as *siderosis*; of carbonaceous particles as *anthracosis*; of cotton particles as *byssinosis*. Other uncouth words have been suggested for different morbid varieties of the same general kind. To multiply such words is undesirable; whatever the kind of dust, the consequences of its presence are, pathologically speaking, substantially the same.

Besides the form we have to recognise other differences of inhaled particles, as, for instance, the density and the chemical qualities which affect their pathological influence. This last, in like manner, is modified by the solubility, miscibility with fluids, and cohesiveness of the dust. A very soluble dust will be got rid of by speedy absorption; a miscible one, like flour and other amylaceous substances, will collect in tenacious masses and obstruct the bronchial tubes; the fine dust of hydrate of lime, unless highly caustic and breathed freely, seems more or less to be disposed of in the parenchyma by absorption and the influences of secretions upon it; whilst that of lime salts—for instance, the carbonate in the shape of chalk, and the sulphate in the form of alabaster in fine powder—may be breathed with impunity for long periods of time.

In numerous cases mixed dusts are encountered; as, for example, in the Sheffield and needle-making trades, where siliceous and metallic dust is intermingled in various proportions. In such instances this circumstance of admixture is presumably attended by some variations in the consequent symptoms. Yet even in this matter the form and dimensions of the scattered particles play a more important part than chemical constitution.

Again, speaking generally, the ill results of dust are more serious the greater its departure from organic tissue in character.

When dust which has entered the respiratory system has reached the alveoli it attaches itself in the first place to the walls, but sooner or later penetrates them. The particles are borne along by mucous cells, which, being destitute of walls, envelop them after the fashion of amœbæ; but the foreign bodies soon prove a source of irritation, which in the case of the more irritant kind advances to inflammation of low intensity, but sufficient to induce an outpour of some lymph and the generation of granular matter and exudation cells. The first effects of the foreign matter are the detachment of the normal epithelial cells of the alveoli and an attendant thickening. The accession of the new material within the alveolar cells causes their distension and functional derangement, and presently, by the action of the exuded fibrin, their obliteration. When a group of such altered cells is formed, the next phenomenon is the production of a piece of more or less solidified lung tissue, manifested in the form of a granule. The multiplication and cohesion of such granules, and the gradual condensation of the fibrin, transform the piece of lung into the fibroid texture we know as pneumoconiosis; whilst the transformation is proceeding the blood-vessels become thickened, blocked, and impervious, and the compressed lymphatics are merged in the morbid growth.

In their general features these structural changes, though chiefly peribronchial, resemble the early stage of croupous pneumonia; but their development is so slow and inactive that no marked fever attends them. However, they possess the faculty of extension; the mucous and inflammatory cells make their way along the adjoining connective tissue, and fibrosis continues to spread in the form of fibrous bands, veins, or streaks.

On section of the condensed portions of fibrotic lung the surface commonly exhibits numerous raised points which give it a coarsely granulated appearance. Most of these points, if closely examined, will be found to be small bronchial tubes, cut across, and thickened and obstructed by secretions and, frequently, by a yellowish matter, suggestive of caseation, which imparts a speckled aspect to the specimen (1254, 1255, 1256). The occlusion in its turn embarrasses the lung yet more, and retrograde structural changes are precipitated.

The morbid phenomena just described are those of dust of distinctly irritating qualities; but when the dust is of a more innocent kind, they are considerably modified. For example, where the dust is organic, but devoid of acrid qualities, as in the cases of the flour of wheat and other cereals, and of like mild substances, the primary irritation is of small account; the dust then operates chiefly as an obstructive agent, clogging the respiratory passages; if any inflammation be present it is very slow. Yet this clogging cannot go on without disabling the air-cells by pressure and otherwise. Soot and very fine charcoal, too, disturb the respiratory organs and their functions to a still less degree; and, though very widely diffused, and causing, it may be, much dyspnœa, they produce no well-marked signs or symptoms of inflammatory action and condensation.

The intimate pathological processes associated with anthracosis (coal-dust condensation) differ considerably from those dependent upon the denser dusts of stone and metal, as is indicated by their morbid anatomy. Nevertheless, indurated lobes are met with, and pulmonary tissue may be so permeated by fine coal-dust as to be rendered friable, and to exude on section and pressure a black, inky fluid; moreover, the expectoration at the same time gets similarly coloured, and acquires the name of "black spit." In some such cases the production of fibrous tissue gives place to a process of disintegration; the pervading dust being, we may suppose, so considerable in quantity as to destroy the vascular supply and lead to a sort of necrosis (1274). In other words, the dusty irritant fails to arouse active inflammation and fibrinous exudation. Examples of the extreme forms of the disease are at the present time unknown, or almost unknown, in English mining districts, thanks to the improved ventilation of mines and the operation of the Mining Acts which limit the age of children admitted to pitwork; for the same reasons the inky expectoration, known in past days as "black spit," is correspondingly rare. It is generally true, indeed, of all dusty occupations, that indurated impervious lungs are becoming rare as sanitary construction is improved, as appliances are used in factories to convey away and disperse the dust, and as sanitary laws and observances are better observed.

The pneumoconiosis due to the coarser dusts which arise from the operations of stone-dressing and quarrying present some peculiarities attributable to the form and composition of the particles. The sawing and polishing of Aberdeen granite appear to be unattended by severe lung irritation; whilst, on the other hand, working with Edinburgh building-stone proves most injurious, and kills the workers by fibroid phthisis in relatively large numbers.

Another peculiarity of the dust of stone is that it tends to collect in masses, forming concretions (pneumoliths) which, by producing softening and ulceration around them, give rise to cavities with soft walls, though now and again lined by membrane. This state of things occasionally ends in the detachment and expulsion of the concretion, as it penetrates into a bronchus of sufficient calibre. Another feature in the lungs of stone-workers is that a yellowish or grayish hue replaces more or less the blackness seen in most other workers (1276, 1285, 1286).

Dust often reaches the pleura and lines its pulmonic surface, imparting a more or less black colour to it, and at other times collecting in nodules upon it. Its transference is effected by the lymphatics.

The ultimate goal of much of the dust inhaled is the bronchial glands, where it becomes imprisoned, causing enlargement and possible suppuration of those organs, which acquire a black colour, often of great depth.

Occasionally the enlarged and softening glands adhere to a contiguous organ, as, for example, to the œsophagus; this may be followed by ulceration through its coats, and the contents of the abscess thus discharged.

Not only do differences of dusts, in their origin, form, and composition, modify the form of the lung diseases which are set up, but they variously affect the health of the sufferers; and as the various conditions under which labour is carried on have also their respective influences, it becomes evident that the symptoms and pathology of pneumoconiosis must exhibit a great variety. Intemperate habits in the patient, for example, precipitate the onset of the lesion, and accelerate its course.

Further, no doubt exists that persons of any age who inherit chest weakness, if they follow a dusty trade, are more liable to suffer from pneumoconiosis. One practical lesson is that such persons should not be thus employed. This inference points to the utility of certifying factory surgeons to pronounce upon the comparative fitness of all who engage in such work, especially of children and youths, and to the care they should exercise in following their occupation.

Again, the lungs of very young children, and of those born of parents with thoracic lesions and damaged constitution, suffer more readily from exposure to dusty occupations, and to heat, moisture, and confined or vitiated air, their too frequent accompaniments.

Associated lesions are found with the fibrotic change in the shape of emphysema, bronchiectasis, cell-collapse, and cavities; morbid events which are described and explained in other parts of this chapter: the first-named change is for the most part vicarious or complementary. A specimen of this vicarious emphysema is No. 1276. The emphysema is not confined to the circumference of solidified segments of lungs, but occurs elsewhere, particularly along the free anterior border; here the dilated cells are much larger, and the septa between them frequently broken through.

Bronchial dilatation or bronchiectasis [*vide* chapter on "Bronchiectasis" in this volume] is usually but not invariably associated with fibroid disease. As the bronchi are the first to suffer from the inhaled dust we might well expect them to be prominent in their lesions. A primary change, indeed, does occur in them in the shape of thickening of their walls, with loss of elasticity, due to the inflammatory action proceeding in and around them. Moreover, whilst losing elasticity they get plugged and distended by mucus and inflammatory products; and the growing fibrous tissue about them, in course of its contraction, drags on their walls, and causes irregularity of form and disturbance of position.

In this view of the causal relations of bronchiectasis we have the support of Dr. Coats of Glasgow and of Professor Hamilton of Aberdeen; but the former contends that both it and the formation of cavities are consequences and not causes of pneumoconiosis. The latter puts forward the following mechanical hypothesis: that "as the chest wall forms a comparatively fixed point to which the shrinking lung tissue also is attached by means of pleural adhesion, and as the tissue also is attached to the walls of the bronchi, the result of the shrinking will be that these two points will be approximated, the chest wall drawn in, and the bronchial wall drawn out. The latter, however, being the more yielding structure, will be more affected than the former. In this way we have the formation of cavities by bronchiectasis. Such cavities have for the most part well-defined walls, and are directly continuous with bronchial tubes of which they are flask-like dilatations. It is to be remembered that the primary process, involving as it does the smaller bronchi, leaves all but these capable of dilatation."

This explanation may hold good in some instances, but cases are not uncommon where no connection by bands with the chest wall exists, and others where cavities occur in crepitant lung tissue.

Pursuing his criticism of Professor Hamilton's hypothesis, Dr. Coats affirms that in some cases cavities arise by accumulation of the secretions and inflammatory products behind an occlusion of a bronchial tube. He believes also "that cavities form by bronchial dilatation by a similar process to that which leads to emphysema without any primary disease of their wall. The dilatation, in fact, is complementary to the shrinking which has taken place in some part of the lung." Continuing these observations, he cites a case of "congenital non-inflation of the lung in which the bronchi had become converted into a series of sacs. Here the non-inflation without any active disease, implying as the chést enlarged an excessive distensible force acting on the bronchi, caused a general bronchiectasis. In a similar way in fibroid phthisis, we may have bronchiectasis and emphysema in an otherwise sound part of the lung, in consequence of shrinking in another part" (p. 124). These secondary changes are features so well marked in the later stages of pneumoconiosis as to call for our attention; but for further details as to the forms and other varieties of bronchiectasis the reader is referred to the special chapter on this disease (*vide* p. 53).

Cavities, not of bronchial origin, may perhaps arise in a portion of pulmonary tissue deprived of function and nutrient vascular supply; as, for instance, in a mass of condensed tissue, whether from fibrosis or from collapse. Again, the presence of a concretion may operate as a cause of softening of the tissue around it, when the cavity may contain a "pneumolith," or only pus and detritus should the stone have ulcerated into a bronchus and made its escape outward (1278, 1279). This phenomenon is most common in the case of coarse mineral dust as found among stone-workers. I have already remarked on the almost constant association of pleuritic thickening with pneumoconiosis, but expressed my dissent from the opinion of Sir A. Clark as to the direct causal relation between the two lesions. For, according to that opinion, fibroid lesion is the result of growth from without inwards. This, indeed, does take place in some instances, as seems to be illustrated by a case described by Clark; but when fibroid lung is due to dust inhalation, the development of fibrous tissue passes from within outwards, and fibrous bands do not extend from the pleura (1282). There seems good reason, moreover, for supposing that a primary inflammatory process is not universally necessary to the development of fibroid mischief; but that any condition which destroys or suspends pulmonary action for a long time, by obstructing access of air and blood-supply, will lead to lung collapse and infiltration, and eventually to fibroid degeneration. In a word, loss of function is a prelude to fibrous degeneration; and Rindfleisch surmises that obstruction of bronchi by accumulated cells and mucus will cause collapse of air-cells in the rear, and ultimately fibroid degeneration.

The notion that a constitutional proclivity lies at the root of pulmonary fibrosis receives some support from Clark's own statement; namely, that albuminuria is commonly associated with it. As regards the unilateral interstitial pneumonia this may be true, but as regards pneumoconiosis, in almost all the cases examined no albumin was found. Albuminuria is, of course, not unknown in such cases, but its occurrence is in no higher ratio than among an equal number of persons suffering from miscellaneous maladies. The concurrence of pneumoconiosis with pulmonary phthisis is not infrequent, but there is not pathological identity between the two maladies. Irritation may be a common starting-point in both; but, whereas in tuberculous lesion the apparent cause is the existence of tubercles and of bacilli, and the prevailing tendency is to soften, break down, or ulcerate, in fibroid disease, on the contrary, there is a chronic inflammation with fibrinous products which tend to contract and transform lung tissue into a hardened mass unfavourable to tuberculous extension and softening. For it is known that a piece of fibrous tissue in a softening tuberculous mass operates as an obstacle to the disintegrating process. Nevertheless, experience proves that the one lesion is often engrafted upon the other and may replace it.

Diagnosis.—Dust-phthisis and pulmonary phthisis have been, and, indeed, still are very frequently confounded. The pulmonary fibrosis of metal-grinders, of stone-workers, of potters, miners, and some other artisans is popularly known as consumption or phthisis, and its victims are entered in death registers as dying of consumption. As already shown, the inflammatory phenomena of deposited tubercle and of fibrosis affecting the pulmonary organs diverge at an early stage; the thickened alveoli and their contents suffer a degenerative process, ending in the former case in softening and ulceration; whilst in the latter an abnormal development of fibrous tissue takes place rather prohibitive of ulceration, but directly productive of condensation and shrinking.

However, this differential feature must not be pushed too far; for, as already asserted, a tuberculous lung presents more or less condensation from excessive development of fibrous tissue, especially where life has been greatly prolonged, and opportunity given for the conservative agency of the fibrinous effusion to advance at a greater rate than that attained by the disintegrating action of tubercle (1302, 1310). A very important preliminary part of the diagnosis is to ascertain the presence or absence of tubercle in the history of the patient's family, or of any organ of his own body. The nature of the work followed, and the conditions and circumstances of employment, will likewise, of course, be ascertained. A third point of importance is to get a correct account of the onset of the malady, its course and its duration. Pneumoconiosis is compatible for many months with capacity for physical labour and for adequate nutrition; tuberculous phthisis, on the other hand, within an equal period, will reduce the patient to a state of considerable debility and emaciation. Tuberculous phthisis is rather a disease of youth and of early middle life, of constitutional nature, and without obvious causal connection with the breathing of dust; whereas pneumoconiosis is a local lesion of middle life, directly referable to dusty employment, and not associated with any marked constitutional bent. Moreover, it is not only most prevalent in middle life, but it is also almost peculiar to men. Nor is the previous history of dust-disease that of acute or febrile thoracic maladies, such as pneumonia or pleuro-pneumonia, but of smaller ailments, especially of winter coughs recurring year after year, and disappearing in the warmth of summer. Other divergent features are the rarity of laryngeal troubles, the comparative infrequency of diarrhœa, and the little pyrexia and sweating in the dust-produced lesion. Hæmorrhage in notable quantity is an infrequent incident in dust-disease, unless the lesion be provoked by sharp particles as of iron and steel. Green purulent sputa are also less common, though not unknown; for in the far-advanced stage they occur. The sputum for a long period is a whitish frothy mucus, which presently gets lumpy and grayish yellow, and for the most part is in smaller quantity, regard being had to the extent of lesion, than in tuberculous disease; speaking generally, the symptoms of dust-disease are more like those of chronic bronchitis, with which, in fact, it is usually confounded, than of phthisis. Fœtid bronchitis and gangrene are unusual, but not unknown results.

The temperature is less elevated than in phthisis, and hectic fever, if present, less pronounced. In the earlier stage emaciation proceeds and may for a time pass unobserved, for usually the appetite and digestion continue more or less good. Diarrhœa seldom appears.

Again, whilst anæmia and œdema are less prominent, dyspnœa is more so, and often paroxysmal. Dyspnœa is greatly aggravated when pulmonary embarrassment has long existed and secondary cardiac disease been established; and as in chronic bronchitis, the cough, expectoration, and hard breathing are increased on first rising, or on passing out into the cold outer air, and are abated by warm drinks and warm atmosphere.

Phthisis particularly affects the apices of the lung, whereas pneumoconiosis prevails rather in the posterior and inferior segments. Moreover it is distributed in separate patches, and gives rise to a greater or less number of areas of dulness on percussion. Dulness under the clavicles, when found, does not resemble that of tuberculous deposit or of pleurisy; it points rather to absence of respiration and bronchial mischief, and is less accompanied by moist crepitus and râles, or by the "cracked pot" sound of a cavity.

In those instances where pleurisy has played a prominent part the adhesions cause deformity of the chest wall and consequent displacement of the heart. The same event happens where the lung itself has become greatly shrunken or displaced by its fibrous transformation (1274).

Yet, notwithstanding differential features, tuberculous phthisis and pneumoconiosis are separable by a somewhat shadowy line (1263, 1264);

and if the presence or absence of bacilli are to furnish the securest foundation for making the distinction between the two, far more careful and numerous researches are needed than have as yet been made.

The intermingling varieties of fibroid and tuberculous disease are well exhibited by Sir A. Clark, who describes two main forms as properly distinguishable, namely, the tuberculo-fibroid and fibro-tuberculous,—the main difference between the two being the order of succession of tubercle and fibroid; but a further examination of those problems would lead us beyond the limits of a chapter on pneumoconiosis.

Of the many cases placed on record as pneumoconiosis by various observers it remains doubtful whether all are true examples of the disease. Some of them certainly are not, and unfortunately the opportunity for studying the disease is to a great extent denied to the bulk of medical men. Indeed, I apprehend from my study of his able treatise on *Fibroid Diseases of the Lung* that its distinguished author lacked material for a thorough exposition of the morbid consequences of dust-inhalation, apart from tuberculous complication.

Non-tuberculous fibroid disease, indeed, whether due to dust or not, is an uncommon lesion, and it is becoming more uncommon day by day by reason of the advances of hygienic knowledge and of its increasing practical application to those employments wherein dust is an almost necessary accompaniment.

Prognosis and Treatment.—From the account given of pneumoconiosis it is evident that when once established a permanent lesion will remain, which cannot be undone by medicinal treatment. Nevertheless, it is capable of great alleviation; and, if not beyond a certain stage, its symptoms will remain quiescent if the sufferer abandon his dusty occupation. Moreover, it scarcely need be said that whatever sanitary arrangements can be provided, and whatever mechanical contrivances can be invented to obviate the entrance of dust within the chest, so much less severe and less frequent will be the disease.

Being a very chronic malady, it affords ample time and opportunities for hygienic management, and for whatever medicinal treatment can be suggested. The first indication, then, is to withdraw the sufferer from his employment, or to diminish the production and diffusion of dust in the work by mechanical and other devices to secure thorough ventilation of shops. Respirators should be worn, the workmen themselves should carry on the technical details of their calling so as to produce the least amount of dust, and observe the general rules of temperance and health in their way of living. On the part of masters it is an imperative duty to provide healthy workshops with efficient ventilating apparatus and all sanitary arrangements calculated to protect their work-people from the evils of the occupation they are engaged in.

Thus fibrotic patients may live many years, though they must be accounted more or less invalid. At the same time it is to be remembered that the dormant lung affection may be easily aroused into activity by fresh exposure to dust, and become complicated or aggravated by bronchitis, broncho-pneumonia, and pleurisy, by depressing causes such as cold and wet, and by irregular and intemperate habits.

Prognosis becomes highly unfavourable when symptoms arise indicative of the development of tubercle in the already diseased viscus. This, unfortunately, happens not infrequently, and is less to be wondered at when we remember the prevalence of this hereditary disease, which abounds in manufacturing populations. The occurrence of hæmoptysis is thus of bad omen.

Of drugs I have found the iodide of potassium the most useful, sometimes, where additional alkali is needed, combined with bicarbonate or citrate of potash; or, where a spasmodic asthmatic state is present, with ether or the ethereal tincture of lobelia. Where great weakness exists, quinine is useful, and cod-liver oil may, when it can be borne, be administered with great advantage to prevent wasting. Terebinthinate inhalations, such as pinol, facilitate expectoration and relieve cough, for which also vapor conii may be inhaled, or a linctus may be ordered containing a minute dose of morphia with some preparation of squills and tolu. But it is of the first importance to sustain nutrition, to encourage exercise outdoors, and to promote action of the chest muscles by regulated calisthenics. Further instructions in treatment will be found in the other chapters on pulmonary disease in this work.

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PULMONARY ASPERGILLOSIS

Short description.—A destructive disease of the lungs due to their invasion by a fungus, the Aspergillus fumigatus. The disease depends on the inspiration of the spores of the fungus, and occurs chiefly in those whose occupation brings them in contact with infected grain. Clinically the disease presents itself under two forms: (i.) like chronic pulmonary tuberculosis; (ii.) like emphysema and bronchitis.

Besides attacking the lungs primarily the aspergillus may become engrafted on pre-existing pulmonary lesions.

Historical.—Hughes Bennett, in 1842, described the first example of pneumomycosis, in which the sputum and cavities of a phthisical subject were found to contain a fungus. Gairdner, in 1853, showed a specimen of a tuberculous lung which had given rise to pneumothorax, with small circular white areas of fungoid growth on the pleural surface, penetrating very slightly ($\frac{1}{16}$ inch) into the lung substance, and measuring $\frac{1}{8}$ inch in diameter. Rayer eleven years before, in 1842, had met with a very similar case. Bristowe in 1854 recorded the case of a woman who died with signs of chronic bronchitis; in the apex of the left lung there were two communicating vomicæ containing no secretion, but on the septum between them there was a powdery, velvety mass of mycelium; although there was no other evidence of tuberculosis: the vomicæ were regarded as being tuberculous. Virchow, in 1856, gave an account of several cases of aspergillary broncho- and pneumomycosis in patients dying from other diseases.

A number of other observers have recorded cases which, like the preceding cases, were regarded secondary infections of pre-existing pulmonary lesions.

In 1890 Dieulafoy, Chantemesse and Widal described clinically aspergillary pneumomycosis in persons engaged in stuffing and fattening pigeons for the Paris market, and struck out a new line in their view that it is a primary affection.

In 1897 Renon collected all the evidence bearing on the subject in his *Étude sur l'aspergillose chez les animaux et chez l'homme*, to which reference for an exhaustive discussion and account of the whole subject may be made.

At first and for a considerable time the occurrence of aspergillus was supposed to be no more than an accidental invasion of already diseased lung tissue, the fungus being merely saprophytic. Thus in Bristowe's case, although there was no sign of tubercle elsewhere in the lung, the lesions were regarded as tuberculous and not due to the activity of the fungus.

But lately the French school, and especially Renon, whose conclusions are based on extensive experimental research, have successfully argued in favour of primary pneumo-aspergillosis; while in England, Boyce and Arkle and Hinds have within the last few years described cases of the primary affection.

Aspergillary pneumomycosis may therefore be considered under the two heads—(*a*) primary; (*b*) secondary.

It is a difficult question, however, in many instances to settle whether the aspergillary affection be undoubtedly primary, and the cause of morbid lesions in a lung previously healthy; or whether it be a secondary infection only. In former times there was a strong and general impression that aspergillary occupation of the lung is essentially an accidental and secondary phenomenon.

Recently Max Podack has expressed doubts whether cases described as primary by the French observers are in reality of this nature; on the other hand, Renon regards Wheaton's "case primarily of tubercle in which a fungus (aspergillus) grew in the bronchi and lung" as being an example of primary pulmonary aspergillosis. Thus different interpretations are put upon the same case.

Etiology.—Pulmonary aspergillosis is a trade disease in Paris; it occurs in persons whose calling is the artificial feeding of pigeons, and in those who comb and sort hair. The essential factor is the intimate relation to grain infected with the spores of the Aspergillus fumigatus. The pigeon-feeder fills his own mouth with a watery mixture of canary seeds and vetch seeds, and transfers the grain to the pigeon's mouth. Spores of aspergillus attached to the seeds thus get into the trachea and are conducted to the air-vesicles, through the walls of which they easily pass. It is remarkable that the alimentary canal of man seems immune to Aspergillus fumigatus. According to Renon, there are only about ten persons engaged in this trade in Paris.

The hair-sorters employ the flour of rye to enable them to separate the hairs more easily; this process impregnates the atmosphere in which they work with dust, which may contain the aspergillus of the rye flour. The atmosphere of their working-rooms is so poisonous that birds die after being exposed to it for a fortnight.

Aspergillosis is a rare disease; it appears more likely to occur in millers, agricultural labourers, and those brought in contact with grain, than in any other class of the community. Apart from the Paris cases a few sporadic examples of the disease have been recorded.

Pulmonary aspergillosis belongs to a class of lesions which, though comparatively little known, has been more studied in animals than man. The lesions of the class pseudo-tuberculosis are granulomata, and resemble those of true tuberculosis, except in respect of the causal agents, which include bacilli other than those of tubercle, fungi of various kinds, and even worms (*vide* Distomum Ringeri, vol. ii. p. 1027). The close resemblance (to the naked eye) of the lesions of pseudo-tuberculosis to genuine tuberculosis renders it very probable that they are often regarded as such; and that, being rarely recognised, this form of lesion is not so infrequent as our present experience would suggest. Systematic examination of pulmonary lesions might prove that some conditions generally dismissed as tuberculous are in reality pseudo-tuberculous, and are due to quite a different cause. Flexner has recently described the condition of Pseudo-tuberculosis hominis streptotrichia in a man who died with the signs of pulmonary tuberculosis, and whose lungs showed consolidation with early excavation.

The aspergilli are true fungi, and belong to the family Perisporiaceæ, order Ascomycetes. Of the varieties of aspergillus, two, A. fumigatus and A. niger, are parasitic, and produce morbid changes in the human body.

Pulmonary aspergillosis appears to be almost always due to A. fumigatus; A. niger has, it is true, been described in some instances, but Renon throws doubt on the accuracy of the observations, and regards them all as examples of A. fumigatus.

Both varieties have been described as attacking the external auditory meatus, and the skin.

It should be remembered that in order to determine the species cultures are necessary, and that without this no opinion as to the identity of the form of aspergillus is valid.

Aspergillus fumigatus flourishes best at the temperature 37°-40° C., while A. niger grows best at 25° C.; and this might be thought to explain the pathogenetic qualities of A. fumigatus; but in Renon's hands experiments on frogs do not support the simple view that it is merely a matter of the bodily temperature suiting the development of one species and not of the other.

Primary pulmonary aspergillosis.—*Symptoms.*—The clinical features presented by the recorded cases of primary pulmonary aspergillosis may resemble either those of chronic pulmonary tuberculosis or those of emphysema.

When the disease takes the first of these two forms there is recurring hæmoptysis, cough, expectoration becoming green and purulent, and signs first of bronchitis, and later of consolidation at the apex. Furthermore there is elevation of the temperature; and pleurisy may supervene. The resemblance, therefore, to pulmonary tuberculosis is so far exact; but if the sputum be examined, tubercle bacilli are absent, while the mycelium of Aspergillus fumigatus is present. The course of the disease is very slow and prolonged; recovery takes place eventually by expectoration of the aspergillus, but the affected portion of the lung undergoes marked fibrosis.

A patient affected with pulmonary aspergillosis offers a suitable soil for tubercle bacilli, and a secondary infection may take place, the aspergillus disappearing from the sputum and being replaced by tubercle bacilli. Renon and Sargent have recorded a case of primary pulmonary aspergillosis succeeded by tuberculosis, in which eventually both these infections became obsolete: but so much chronic pneumonia resulted that death from failure of the right side of the heart terminated the case. In another and similar case related by Renon the sputum first contained the aspergillus alone; later very scanty traces of it were found, but plenty of tubercle bacilli, and eventually no bacilli or aspergillus, the patient surviving with evidences of chronic pneumonia.

In the emphysematous form the disease may run a rapid course, as in the case recorded by Arkle and Hinds. Hæmoptysis is infrequent, or may not occur at all; there is loss of flesh and strength, frequent cough and severe dyspnœa come on in attacks at night, and suggest spasmodic asthma. The physical signs are chiefly those of emphysema and bronchitis.

Intermediate forms between these two may occur, signs of apical consolidation supervening in the emphysematous varieties; and conversely cases which appeared like chronic phthisis may be marked by attacks of pseudo-asthma.

Morbid anatomy.—The data at our disposal are somewhat scanty, but so far as they go they tend to show that the morbid appearances in the lungs met with in the described cases of aspergillosis differ just as do the lesions of acute and chronic tuberculosis. This difference depends on the resistance offered by the lung tissue to the inroads of the fungus. It will be most convenient to describe the anatomical lesions in connection with the two chief clinical types of the disease to which attention has already been called.

1. In cases where the disease has run a very chronic course, resembling either chronic pulmonary tuberculosis or chronic pneumonia, the aspergillus may either (*a*) still be found on the lung tissue, or (*b*) it may have been entirely removed, and then have left behind it a chronic interstitial pneumonia which eventually proved fatal.

(*a*) Our knowledge of the lesions existing in primary aspergillosis when the aspergillus is still present in the lung tissue is particularly scanty. Renon bases his description on two cases, those of Boyce and Kohn. The lung tissue contains dilated bronchioles leading into cavities in pneumonic areas, in which there are pseudo-tubercles composed of hyphæ so arranged as to resemble actinomycosis. There is much phagocytic reaction in the pneumonic areas, showing that very active resistance had been opposed by the lung tissue to the aspergillary invasion. Renon associates the actinomycotic form adopted by the aspergillus with the active resistance of the tissues, and considers it as an indication of defensive powers on the part of the tissue and of lowered vitality on the part of the aspergillus. Hence this form of pneumo-aspergillosis is called by Renon "abortive." The cavities also contain the aspergillus. The process is essentially the same as that in cases of aspergillosis; its clinical features are those of emphysema, namely, consolidation and destruction of pulmonary tissue; but it is a local process which has become arrested at an earlier stage.

(*b*) In a case of primary pulmonary aspergillosis, described by Renon and Sargent, in which true tuberculosis supervened with disappearance of the aspergillus from the sputum, death took place from failure of the right side of the heart, and examination of the lungs showed chronic pneumonia; but no trace remained either of the aspergillus or of tubercle bacilli.

2. In cases where the symptoms have been those of emphysema and dyspnœa the lungs contain patches of consolidation breaking down into cavities, while there is compensatory emphysema which may be well marked. The lesions in Hind and Arkle's case have some analogies with Tooth's case of acute bronchiolectasis, though in the latter the causation had nothing to do with aspergillosis.

Microscopically the walls of the small bronchi are thickened, and both the lung substance and the alveolar cavities contain the aspergillus mycelium. In places the lung tissue is so disorganised as to be unrecognisable, and there is breaking down of the lung tissue leading to the formation of microscopic cavities. The mycelium is in extremely intimate relation with the lung tissue, and, as it is accompanied by phagocytic reaction, the aspergillary invasion of the lung tissue appears to be the direct cause of the lung lesions, not a merely accidental or post-mortem event.

Since no toxin has been obtained either from the media in which the Aspergillus fumigatus is grown (Kotliar), or from the fungus itself (Renon), it appears probable that the large quantities of the fungus in the lung tissue set up the inflammatory changes by mechanical irritation. The absence of any toxin explains the comparatively mild character of the disease; but it makes it somewhat difficult to understand why A. fumigatus is the chief if not the only variety of aspergillus pathogenetic for pulmonary tissue.

Generalisation of aspergillosis does not occur.

Diagnosis.—The physical signs are not in any way characteristic, and would point to bronchitis and emphysema or to chronic pulmonary tuberculosis. In Wheaton's case there was a growth of the fungus at first white, later black on the tongue and palate. But this is the only help that ordinary methods of physical examination can be expected to supply, and, unfortunately as regards diagnosis, this coexistence of oral and pulmonary aspergillosis is almost unique.

The diagnosis depends on the presence of the fungus in the sputum, and the absence of the tubercle bacillus. In cases where tubercle becomes engrafted on primary pneumo-aspergillosis, both organisms might be found in the sputum; and, unless the patient had been under observation from the beginning when the aspergillus alone was present in the sputum, there would be no means at first of distinguishing the primary form complicated by tubercle from secondary aspergillosis occurring in the last course of pulmonary tuberculosis.

The fungus, derived from dust, is occasionally found in the mouths of healthy persons.

Cultures of the aspergillus in appropriate media, such as Raulin's fluid, and inoculation of animals may be necessary to determine that the form of aspergillus is the pathogenetic Aspergillus fumigatus, and not the other non-pathogenetic varieties, such as Aspergillus niger, glaucus, and so forth. It must be distinguished from the streptothrix form of the bacillus tuberculosis; and, lastly, the lesions must be distinguished from other forms of pseudo-tuberculosis due to different factors such as bacteria, streptothrix, actinomyces, or Distoma Ringeri.

The prognosis of pulmonary aspergillosis is less grave than that of pulmonary tuberculosis, since the lesion is usually much slower, never sets up a general infection comparable to generalised tuberculosis, and tends to undergo a gradual and spontaneous cure. But there are several reservations to this general statement. For, even if the aspergillus disappear, the lesions of chronic interstitial pneumonia may lead to dilatation of the right side of the heart, and so to a fatal result.

It need hardly be said that the development of genuine tuberculosis renders the prognosis much graver.

The prognosis of the emphysematous form does not, from the few recorded examples, appear to be nearly so favourable as that of the more chronic variety which has been likened to chronic tuberculosis.

Treatment.—Although there is no specific remedy for pulmonary aspergillosis, nor any drug that can be employed to kill the fungus outright in this situation, experiments on animals show that iodine, iodide of potassium, and arsenic increase the resistance of the organism to the invasion of Aspergillus fumigatus and inhibit its growth; their employment is therefore reasonable in this disease in man. The general strength should also be improved by good and generous feeding, cod-liver oil, tonics, and fresh air; thus we may guard against secondary infection of tubercle, and assist the tissues in their struggle against the aspergillary infection.

Symptoms should be treated as they arise. When hæmoptysis occurs the treatment is the same as in pulmonary tuberculosis. Attacks of asthma may be relieved by iodide of potassium, tincture of lobelia, and other appropriate remedies; while creasote, terpene, turpentine, may with other drugs be given for bronchitis.

Removal from the poisonous atmosphere is an important essential, both in prophylaxis and in treatment.

When tuberculous infection has taken place, the course of treatment is that of chronic pulmonary tuberculosis.

Secondary pulmonary aspergillosis.—Here the Aspergillus fumigatus develops as a result of the inhalation of its spores; and finds a suitable nidus in lung tissue the resistance of which has been already much lowered by pre-existing disease, or has actually undergone necrosis.

It has been found in the bronchi and in the lung substance. Thus the aspergillus may be engrafted on bronchiectasis of old standing; or may take root on the walls of vomicæ due to tuberculosis; or in the lung under other conditions, such as malignant disease, pulmonary apoplexy, chronic bronchitis, broncho-pneumonia, and gangrene of the lung.

In some of the cases where it has been described as secondary, it may, as already hinted with regard to Dr. Bristowe's case, in reality have been primary.

In cases where there are multiple bronchiectases or vomicæ in the lungs, the absence of the fungus from some of them and its presence in others are strong evidence in favour of the secondary nature.

It is remarkable that in gangrene of the lung associated with the presence of aspergillus there is no fœtor. It seems that the growth of the micro-organisms of putrefaction is prevented by the aspergillus.

The actinomycotic form of the mycelium appears to occur where there is considerable reaction and resistance on the part of the tissues, and it is probable that it does not occur in secondary or terminal aspergillosis.

Clinically speaking, secondary aspergillary pneumomycosis, like thrush in the mouth of adults, is probably a precursor of death, and is not likely to be suspected or discovered unless the mycelium be found in the sputum. It is in fact a terminal complication.

The treatment is that of the primary disease on which the aspergillosis has been engrafted.

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EMPHYSEMA OF THE LUNGS

Definition.—A disease of the lungs characterised by over-distension of the alveoli and atrophy of the alveolar walls.

It has been the custom to describe under this heading two essentially distinct morbid conditions; the one, corresponding in anatomical details to the definition above given, having nothing in common with the other but the name. An account of this latter affection, *interlobular or interstitial emphysema*, will be found at the end of this article.

The description of emphysema of the lungs given by Laennec, accurate though it was as regards both anatomical characters and clinical history, remained incomplete until supplemented by the microscopical researches of Rokitansky and the clear exposition of its pathology which we owe to Sir William Jenner. Our knowledge of the disease has been mainly derived from their writings, and few additions of importance have been made to it in recent years.

Pathogeny.—Various hypotheses have been advanced to explain the origin of emphysema, some of which meet with but little support at the present time. It would serve no useful purpose to enter upon a detailed discussion of the problem, as it is exhaustively dealt with in the original papers of Sir William Jenner, to which reference may be made. It will be sufficient to mention those views which have at any time received considerable support, and to discuss in greater detail that which is now generally adopted.

Primary degeneration hypothesis.—The view that the general cause of emphysema is a primary fatty degeneration of the alveolar walls was first stated by Rainey, and subsequently received support from Villemin. The latter writer describes the changes as beginning in an excessive proliferation of the intercapillary nuclei, followed by secondary fatty degeneration of the nuclei and other structures, the result of pressure upon the capillaries. It is now generally considered that the degenerative changes in the alveolar walls are secondary to the distension of the air-vesicles and interalveolar spaces, and to the diminution in the blood-supply thereby induced.

It is possible, however, that in the form of emphysema met with in old people, primary degenerative changes may play a more important part. Reference will be made to this point subsequently.

Inspiratory hypothesis.—The hypothesis that emphysema is due to distension of the lungs during inspiration was really first advanced by Laennec. He believed that the air drawn into the lung in inspiration was retained, being unable to escape during expiration, owing to the obstruction caused either by catarrhal swelling of the mucous membrane of the bronchi or by accumulation of mucus in the tubes; and that as a consequence the lungs became over-distended with air.

Dr. Gairdner, in 1850, stated the inspiratory hypothesis in a different form. According to his view, some change in the lungs, such as collapse or retrocedent tubercle, leading to a diminution in size in one part, preceded the establishment of emphysema. As the air-vesicles within the area of disease or collapse did not expand during inspiration, an undue strain was thrown upon those in the immediate neighbourhood by the incoming air, and in consequence they became enlarged.

This opinion, as regards the general disease, has been completely displaced by that to be next mentioned; and as an explanation of the conditions found around patches of collapse or of fibroid tubercle—compensatory emphysema—it is believed that the distending force of inspiration, although possibly not without effect, is subordinate to that of forced expiration.

Expiratory hypothesis.—In 1845 Mendelssohn first advanced the opinion that emphysema is produced during a forced expiration. He believed that the air is prevented from escaping from the upper lobes by the compression of the lungs during forced expiration; that consequently the pressure within the lung is increased, and the air-vesicles undergo dilatation. In 1857 Sir William Jenner stated the above-named hypothesis in the following terms: "The lung during expiration is compressed at different parts with different degrees of force. The parietes of the thorax, in consequence of their anatomical constitution, yield to the same force at different parts with various degrees of facility. The chosen seats of emphysema are exactly those parts of the lung which are the least compressed during expiration, and which are situated under those portions of the thoracic parietes that give way the most readily before pressure."

In a footnote to his paper on "Emphysema of the Lungs," in Reynolds' *System of Medicine*, Sir William Jenner stated that he was unacquainted with Mendelssohn's paper when he advanced this hypothesis in 1845; and that, so far as he was aware, the existence of that paper was unknown in this country until 1867, and rarely, if ever, referred to abroad until that date.

Having regard to the above facts, to the singular completeness of Jenner's papers, and to his demonstration of the exact sites of emphysema, we may fairly regard him as having been the first to make known the true mode of origin of the disease.

The increased pressure in the air-passages, which we have seen to be a common antecedent of emphysema, may be induced in various ways.

Cough.—The almost invariable association of some degree of emphysema with chronic bronchitis points to cough as the most frequent cause of the disease. The chest having first been filled with air, the glottis is closed, a violent expiratory effort is made during which the tension within the air-passages is enormously increased, the glottis then relaxes, the air passes rapidly through the narrow orifice, and a cough results. It is the frequent repetition of this act which eventually induces a permanent dilatation of the air-vesicles and interalveolar passages. The effect of the compression of the lungs during a violent expiratory effort, such as that above described, is to drive the air in all directions from the central to the peripheral part of the lungs; the result is the distension of those parts which are least supported. As pointed out by Sir William Jenner, these parts are the apices, the anterior margin of the upper lobes, and the margins of the bases of the lungs. These are the sites of the primary lesions; but, in the course of the enlargement of the thorax which they entail, the relative position of a given area of lung and the chest wall gradually changes, fresh portions being brought into contact with the intercostal spaces, the resisting power of which is less than that of the ribs, and thus in course of time the change may become general throughout the lungs.

Muscular effort.—It is probable that next to cough violent muscular effort is the most common cause of emphysema. The mechanism is as follows:—the lungs having been completely expanded by a deep inspiration, the glottis is closed; any severe and sustained muscular effort with the thorax in this position necessarily subjects the lungs to strong compression, the increase in pressure within the air-passages being most effectual in distending the lung in those situations where the organ meets with least support. Straining in constipation may have the same effect.

Further reference will be made to causes of over-distension in describing the etiological factors of the disease.

It will be convenient here to refer to those conditions of a temporary nature which lead to over-distension of the air-vesicles. In such cases when the cause is removed the effect may disappear; but whether it does so or not depends upon the duration of the exciting cause and the integrity of the elastic tissue of the lung.

The best example which can be given of this temporary over-distension of the lungs is the condition observed during a paroxysm of asthma. At the height of the attack the lungs may be found distended with air to a degree equal to that present in the most advanced cases of emphysema; but when the attack has passed off, the organs may return to their previous size. It is rare, however, to meet with patients whose asthma is of long standing who are not also the subjects of emphysema.

The mechanism by which this state of over-distension is produced appears to be a matter of doubt; the explanations vary with the hypotheses concerning the cause of the asthmatic paroxysm. If the hypothesis of a spasm either of the diaphragm or of the muscles of inspiration be held, there is little difficulty in understanding why the chest is in a condition of extreme inspiratory distension; if, on the other hand, we reject both these views and accept that now generally received, namely, that the asthmatic paroxysm is due to bronchial obstruction, the result either of a spasm of the muscular fibres of the bronchi or of a fluxionary hyperæmia of the bronchial mucous membrane, the explanation of its mode of occurrence is not quite so obvious.

It is, as a rule, gradual in onset and also in decline, and is apparently brought about in the following manner:—

(i.) The bronchial obstruction induces increased inspiratory effort.

(ii.) The entering air passes the obstruction with difficulty, but the gradually increasing prolongation and force of the expiratory act shows that the air meets with still greater difficulty in escaping from the lungs.

(iii.) Expiration, although prolonged, is not sufficiently so to produce an equilibrium between the incoming and outgoing air; a fractional addition is therefore made to the residual air by each completed act of respiration, and in time the lungs become over-distended.

It may be objected that, as the force of expiration is greater than that of inspiration, the obstruction should be more easily overcome by the outgoing than by the incoming current of air; but it would appear that experience teaches us to rely upon forced inspiratory efforts to remedy a defective aeration of the blood, whereas the condition really requires for its relief forced efforts limited to the period of expiration.

Another possible factor in the production of this state of extreme distension is the compression of the smaller bronchi by the distended alveoli, an effect necessarily more felt during expiration.

Other causes of temporary over-distension of the lungs are laryngeal obstruction, from whatever cause arising, whooping-cough, acute bronchitis in children, and severe muscular strain.

Causation.—*Age.*—It is a matter of common experience that the disease may be met with at any age. Some of the most marked examples are seen in young children. The atrophic form of the affection (see Varieties of Emphysema, p. 269) is most often met with in old people.

Sex.—Men are naturally more subject to the disease than women, as they are more exposed to the conditions which favour its development.

Occupation.—Any occupation involving severe muscular effort, especially if performed with the lungs distended and the glottis closed, tends to produce emphysema. In all such efforts the chest is forcibly compressed by muscular contraction, and the act is equivalent to one of forced expiration. The classical example of an occupation involving the latter condition is that of a cornet-player. Smiths, hammermen, and porters engaged in lifting heavy weights are all liable to emphysema. Omnibus and cab drivers, and all persons whose occupations involve exposure to inclement weather, are prone to attacks of bronchitis, whence comes emphysema. The inhalation of dust, a condition almost inseparable from many occupations, necessarily induces catarrh of the bronchi; upon this cough and emphysema follow.

Diseases such as whooping-cough and chronic bronchitis present the conditions essential to the production of emphysema to the fullest extent. The violent respiratory acts in many forms of dyspnœa may lead to extreme over-distension of the lungs, which may be either temporary or permanent. The same is true in cases of extensive collapse of the lungs as regards those parts into which the air is free to enter. The mode of production of emphysema in asthma and allied conditions has been considered above.

The onset of emphysema will naturally be favoured by any conditions, such as chronic congestion from valvular disease and chronic bronchitis, which tend to diminish the natural elasticity of the lungs. Advancing age is a factor which operates in a similar manner.

Hereditary predisposition.—It has been suggested that there exists in some individuals and families an hereditary tendency to the disease; but this view is rarely insisted upon at the present time. Various observers have investigated this matter, the result being the supposed discovery of the hereditary tendency in a proportion of cases varying from 12 per cent (Lebert) to about 60 per cent (Fuller, Jackson) in adults, and 100 per cent of cases in children (Jackson). It is probably true, as pointed out by Sir William Jenner, that the tendency is not to the disease itself, but to conditions which dispose to it.

Although, however, we may not admit heredity in its most absolute sense to be a cause of emphysema, it does not follow that what, in the absence of precise knowledge, we call the "constitution" of the patient has no influence in determining its occurrence. The tone of muscle and its capacity for energy vary enormously in different persons, though no structural differences can be demonstrated; and the same may be true of the elastic tissues. That such is the case is certainly possible, and in my opinion probable; if so, the occurrence of dilatation of the pulmonary alveoli may well be brought about in certain persons by a degree of increased pressure within the air-passages, such as accompanies ordinary straining efforts, which we are not accustomed to regard as adequate to the production of emphysema, and which, in persons of firmer fibre, are not adequate.

All who have studied the subject of emphysema from a clinical standpoint must have met with cases in which the ordinary proximate causes of the disease seemed to be absent. In many of these the absence has, it is true, been but apparent, for it is difficult to realise how slightly a chronic winter cough impresses itself upon the memory of some patients; hospital patients, indeed, rarely mention such an ailment unless directly questioned about it. But due weight having been given to this source of error, there undoubtedly remains a certain small proportion of cases in which no adequate exciting cause can be discovered. This lack of resisting power on the part of the elastic tissues of the lung may certainly be acquired, it may possibly be inherited, and is probably a common result of the degenerative processes incidental to advanced age. A case recorded by Hugner proves clearly that after recovery from an attack of pneumonia emphysema of the affected part may ensue upon the resumption of an occupation, such as that of a cornet-player, which favours the occurrence of the disease, but which had been previously followed without injury to the lungs.

Normal anatomy of a pulmonary lobule.—A lobule of the lung may be regarded as a lung in miniature; a clear idea of the structure of a single lobule will therefore enable us without much effort to construct the whole organ.

Each lobule, more or less cone-shaped, is surrounded by areolar tissue; at its apex the lobular bronchus, the blood-vessels, lymphatics, and nerves unite to constitute it. The bronchus, after a short course within the lobule, divides and subdivides, with at first but slight diminution in size, forming passages which are termed the interalveolar or intervesicular passages. The course of the bronchus is at first fairly straight, but as the divisions increase in number and diminish in size the direction constantly changes. As the alveolar passages approach the surface of the lobule they cease to diminish in size. Each passage beyond the final division ends in a blind extremity, which, if not dilated, often appears to be so, from the fact, above stated, that the passages do not diminish in diameter. In some cases, however, the ends of the alveolar passages are really dilated, and from this appearance the name "infundibula" has been applied to them; but a distinctive name is scarcely necessary. As the bronchus enters the lobule rounded orifices appear upon its walls. These are the openings of the alveoli, which may be regarded as the radicles of the bronchial tree. They are at first but few in number, but gradually increase. As the air-channel passes onwards through the lobule, and the interalveolar passages are formed, their walls become more and more thickly studied with the orifices of the air-vesicles, until, by the time the surface of the lobule is reached, the blind ends of the passages are found to consist entirely of the orifices of these small recesses.

From the foregoing description it will be seen that the air-vesicles of the terminal passages open into a common space, adjacent vesicles being separated by incomplete partitions; and that all the air-cells of a single lobule are, to a considerable extent, confluent one with another. Adjacent interalveolar passages are separated by partitions formed at the site of branching of the air-vessels.

The interalveolar passages and their terminations are chiefly composed of unstriped muscular fibres, arranged circularly, and supported by a delicate fibroid tissue mingled with elastic fibres. The walls of the air-vessels consist of a delicate membrane crossed by a network of elastic fibres.

The capillaries on the terminal passages are covered by epithelium only on the surface looking towards the cavity; those in the septa project into the cavities on either side.

Morbid anatomy.—The primary lesion in emphysema consists in an enlargement of the terminal interalveolar passages, which increase in size at the expense of the alveoli opening into them. Sometimes, however, the alveoli appear to be the first to undergo dilatation. In any case the effect is, by pressure and stretching, to diminish the blood-supply to the epithelial and vascular structures in their walls. The alveolar epithelium undergoes fatty degeneration, the granules being aggregated round the remains of the nuclei. The septa between adjacent alveoli are reduced to small projections by a gradual process of wasting; subsequently the partitions between neighbouring alveolar passages are perforated, and they become fused into rounded spaces, the size of which tends to increase with the continued operation of the immediate cause of the disease. It is obvious that this process must be accompanied by a great destruction of the pulmonary capillaries, an important factor in determining some of the effects of the disease. According to Rindfleisch, wide communications are formed between the pulmonary artery and the pulmonary and bronchial veins, thus relieving the tension of the former vessel, but allowing the blood to pass through the lungs without undergoing proper aeration.

Varieties of emphysema.—Certain varieties of the disease may be recognised both clinically and pathologically; the morbid changes by which they are characterised will now be considered.

Large-lunged emphysema (Chronic hypertrophic emphysema).—The objection to the term hypertrophous as applied to this condition is that its use connotes increased functional activity, whereas in emphysema the opposite condition prevails. The name here adopted, which was first suggested by Jenner, appears preferable, as it describes the condition and involves no hypothesis.

When the thorax is opened the lungs not only fail to collapse, but remain fully distended, and, when the smaller bronchi have been obstructed from inflammation, may even bulge forward. The apices fill the supraclavicular regions, and the enlarged anterior margins may be in contact beneath the whole length of the sternum, the precordial area being occupied by the distended auricular process of the left upper lobe. The diaphragm is depressed owing to the permanently inflated condition of the lungs. After removal, when the organs are held with the base upwards, the distended and rounded edges of the lower lobes form the sides of a deep cup.

The lungs in emphysema were likened by Laennec to a pillow of down, and the simile can scarcely be improved upon. They are soft and non-crepitant; when compressed a deep pit forms and remains. They are pale gray in colour, and are marked by black pigment, scattered over the surface in lines and spots, the lines in some cases mapping out the lobules. On close inspection the superficial portions have the appearance of a very fine froth, consisting of very minute air-bubbles covered by the pleura. This is rendered more obvious by the use of a hand-lens.

In some cases large rounded air-containing bullæ are present, usually along the anterior margin of the upper lobes or around the bases, but they may be absent when the disease is advanced and widely disseminated. Some are attached to the lung by a narrow peduncle only, the auricular process of the left upper lobe being a common site of this particular lesion. They collapse when opened, and delicate fibrous bands, the remains of alveolar septa and obliterated vessels, may then be found crossing the interior.

These two forms, the "local" or "bullous" and the "general," are too frequently associated to justify a separation in nomenclature; but it is important to bear them in mind, as will appear when we come to consider the physical signs of the disease.

On section the lungs are bloodless and dry, except perhaps at the bases, where œdema may be present. This, however, pertains more to some complication, such as bronchitis or cardiac failure, and is no necessary effect of the disease.

If the section be made from the extreme posterior margin forwards, the portion of the lung which occupies the hollow beside the spine will often be found in an advanced condition of emphysema; large spaces being present beneath the pleura, and extending for perhaps half an inch or more into the lung.

The smaller bronchi are in some cases dilated to a slight degree, but bronchiectasis is by no means frequently associated with emphysema.

Atheroma of the pulmonary artery is commonly present, and in advanced cases patches may be found throughout the vessel, not even the smaller branches escaping: it is a result of the increased strain on the walls of the vessel from the obstruction to the passage of the blood through the lungs. There is very often a complete absence of pleural adhesions, a condition rarely observed in adults unless they are subjects of emphysema.

Small-lunged emphysema; Senile atrophic emphysema (syn., *Senile atrophy of the lungs*).—The most striking clinical and pathological characteristics of this condition of the lungs are indicated by its name. It appears to be primarily an atrophic change, incidental to advanced age, and shared by the lungs equally with the other organs of the body. Its title to be considered either as a substantive disease of the lungs or as a distinct variety of emphysema is doubtful. It never occurs apart from a general condition of atrophy; and the slight degree of emphysema which accompanies it is probably induced by the cough of a bronchial catarrh, from which the very aged are rarely quite free. It is, however, convenient and in accordance with custom to describe it as a variety of emphysema. The subjects of senile emphysema present a wasted, shrivelled, and withered-up appearance: the thorax is rigid, the space within is small, the lower ribs are almost in contact and very obliquely placed. On opening the chest the uncovered area of the heart is not diminished, it may even be enlarged; the lungs readily collapse, falling back towards the spine; they are smaller than normal, deeply pigmented, almost black in colour; light, dry, and easily compressible. On section they present a coarsely reticulated structure. The vesicles are enlarged by a process of fusion, the result of wasting of the septa; and this change may in places be so advanced as to involve adjacent lobules. Large bullæ are rare, but the margins are in some cases much dilated. The bronchi are thin-walled, and have undergone dilatation; the lining membrane is commonly inflamed, and the tubes contain puriform fluid. Collapse and œdema are often present, and are generally most marked on the posterior aspect of the lower lobes.

Local emphysema; Compensatory emphysema.—This form of the disease is invariably secondary to some pulmonary lesion, most commonly to tuberculosis which has undergone either complete or partial arrest. In the presence of a contracting lesion within the lungs—for instance, a cavity or an area of fibroid tuberculosis—either the surrounding tissue becomes emphysematous or the pleura thickened; the result being determined by the nature, site and extent of the lesion. In the case of a lesion situated close to the surface, if the lung intervening between it and the pleura be condensed, airless, and incapable of expansion, the visceral and parietal layers of the pleura, partially united by fine fibrous bands, tend to become separated. The space is at first filled with yellow serous exudation, which ultimately undergoes transformation into a thickened fibroid tissue almost cartilaginous in density. The apex of the lung, in cases of very chronic pulmonary tuberculosis, when the upper lobe is almost completely occupied by a contracted thick-walled cavity, shows such a thickening of the pleura as is here described. If, on the other hand, the lung tissue around the lesion is not the seat of such advanced changes, and still admits of the entrance of air, the surface vesicles enlarge, coalesce, and form bullæ, sometimes of considerable size. Such a condition is commonly seen at the apex of the lung, and is a certain guide to a contracted lesion within. The surface may be scarred and puckered, and on section dense pigmented fibrous bands are seen surrounding old fibrous, caseous, or calcareous lesions, and extending into the neighbouring emphysematous tissue. The vessels and bronchi in such an area are usually obliterated, but on its confines the latter may be found dilated.

Another common site of local emphysema is the posterior and upper part of the lower lobe. Here the change is secondary to a contracting lesion, usually a cavity, at the apex of the lung; and may occupy a considerable area. In one such case observed by myself the posterior aspect of the contracted upper lobe was completely covered by the upper part of the lower lobe. No bullæ are formed, but on section a coarsely-reticulated structure is seen, replacing the normal tissue and reaching downwards along the posterior aspect of the lobe.

In cases of fibroid transformation of tubercle the densely pigmented contracting fibrous nodules are often found embedded in emphysematous lung; the whole presenting appearances which show unmistakably that the fibrosis has preceded the emphysema.

Acute vesicular emphysema.—The definition of the disease given at the head of this article does not include a lesion consisting merely in an over-distension of healthy alveoli, such as is present in the above-named condition. Atrophy of the alveolar walls is an essential part of the morbid anatomy of emphysema, and in its absence we cannot recognise acute vesicular emphysema as a true variety of the disease. It is sometimes found after death from acute bronchitis, or from asphyxia, which had been accompanied by violent inspiratory efforts; or when, from collapse or other cause, the air has been prevented from entering portions of the lung, thus throwing an increased strain upon the alveoli of other parts.

It may be demonstrated, however, by physical examination that a similar condition is present in cases which are not fatal; and also that after a time the lungs return to their normal size, a proof of the absence of structural damage.

The lung in such a condition of over-distension is large and pale, and with a hand-lens the increase in size of the surface alveoli can be readily seen.

Lesions associated with emphysema.—*Lungs.*—Although, in the majority of cases, bronchitis and emphysema stand related to one another as cause and effect, it is nevertheless true that when emphysema has become established it increases the tendency to bronchitis.

The over-distended air-vesicles compress and obstruct the capillaries and impede the circulation through the pulmonary and bronchial vessels. The bronchial mucous membrane becomes congested, and the condition thus established greatly increases the liability to inflammatory attacks. Rupture of dilated vesicles may lead to pneumothorax; but if the pleura overlying the site of rupture remains intact, interlobular emphysema results. Death is rarely due to pneumothorax so caused, but one such case has been observed by myself, and others are on record.

Bronchi.—As already described, the bronchi are often found obliterated and forming thin fibrous bands in large emphysematous bullæ; they are, however, occasionally, but not commonly, found dilated to a moderate degree in less advanced cases of general emphysema, and more often in localised emphysema. In the atrophic form the bronchial walls are usually thin; in other forms they may be somewhat thickened, as may also be the walls of the vesicles and interalveolar passages.

Heart.—The obstruction to the flow of blood through the capillaries of the lungs naturally increases the pressure within the pulmonary artery and requires a more forcible contraction of the right ventricle. This leads to hypertrophy of the ventricle, and thus for a time equilibrium may be restored. But when, from any cause, the structural integrity of the new muscular tissue is impaired, particularly if at the same time greater stress is thrown upon the right ventricle, dilatation follows, the tricuspid orifice enlarges, and the valve becomes incompetent.

The right auricle, probably already somewhat enlarged, now undergoes still further dilatation, and the superior and inferior venæ cavæ are similarly affected. Congestion of all the organs which are drained by the systemic veins necessarily follows. The portal system may become involved at a later period. This sequence of events is not uncommonly initiated by an attack of bronchitis.

The dilatation and hypertrophy of the right ventricle, including the conus arteriosus—for the latter is always involved—are usually found on autopsy to be associated with similar but less advanced changes in the left ventricle; a result probably due, at least in part, to their intimate association both in structure and functional activity.

Degenerative changes are often observed in the heart in emphysematous subjects, and the impaired nutrition of the muscular walls may be due to obstruction to the return of blood by the coronary veins.

As a result of the enlargement of the lung and the permanently depressed state of the diaphragm, the position of the heart becomes altered. It lies lower in the chest, and its axis is more nearly horizontal. The front of the heart is formed entirely by the enlarged right ventricle and auricle. The altered position and size of the organ account for the pulsation commonly observed in the epigastrium in well-marked cases of emphysema; but of these two factors the change of position is the more important.

Secondary changes of a fibroid character are not infrequently found in the tricuspid and mitral valves; and, more rarely, in the aortic valve also.

Liver.—The changes in the liver resulting from chronic venous congestion are too well known to require complete description. The organ is enlarged and the hepatic veins are dilated. The section presents the "nutmeg" character, and there is some degree of induration; but emphysema alone is as powerless as chronic mitral disease to produce a true cirrhosis.

The kidneys may be enlarged and cyanotic, but in a considerable proportion of cases they are granular from the presence of chronic interstitial nephritis, a disease with which emphysema is not uncommonly associated. The *spleen* is as a rule enlarged and hard, but its condition varies.

Chronic venous congestion of the *stomach* may give rise to catarrh and hæmorrhage into the mucous membrane. The *brain* also shows evidence of venous congestion.

As considerable differences exist in the symptoms and physical signs which characterise the various forms of emphysema, it is necessary to describe them under their respective headings.

Symptoms of large-lunged emphysema.—The symptoms strictly referable to emphysema are very few, the condition, apart from its complications, being one of which patients have little or no knowledge, and one of which therefore they rarely complain.

Dyspnœa is the most important symptom, but even this is seldom mentioned until it has become somewhat urgent: it is in proportion to the extent of the disease. At first slight, and only experienced on exertion, especially on walking uphill, it may gradually increase, until in the end not only exercise, but even movement becomes impossible.

It is always much increased during an intercurrent attack of bronchitis, and tends, as the disease progresses, to occur in paroxysms, a condition to which the term "bronchial asthma" is usually applied. The asthmatic element in such cases may either arise directly from the emphysema—the more common order—or the emphysema may be a consequence of asthma. The difficulty of breathing is increased by anything which interferes with the descent of the diaphragm, such as flatulent distension of the stomach or intestines, stooping, or sitting in a low chair after a meal. Orthopnœa follows as the disease progresses, the patient sleeping either propped up with pillows or in a sitting position.

Cyanosis may be considerable, even whilst the patient is still capable of movement—a combination rarely met with except in this disease.

Cough.—Sufferers from emphysema are rarely free from cough for long intervals, although cough is, strictly speaking, due rather to the condition of the bronchi than to the change in the lungs. It is loud, harsh and wheezing, and, like the dyspnœa, may occur in paroxysms. It is always more troublesome in the winter, and particularly so when the weather is cold and damp, or when fog is present.

Expectoration.—Emphysema does not of itself give rise to secretion, but it is by no means uncommon for patients to expectorate a small quantity of mucus to which the descriptive word "pearly" is usually applied. When bronchitis occurs, expectoration becomes profuse, and passes through the various phases usual in this disease.

Hæmoptysis, although an unusual complication of emphysema, may occur, and may even prove fatal. It is generally small in amount. Having regard to the frequent association of atheroma of the pulmonary artery with emphysema, it is perhaps surprising that rupture does not more often happen.

The *appetite* is often poor; complaint may be made of flatulent distension of the stomach and intestines, and constipation is not uncommon.

The deficient aeration of the blood may give rise to drowsiness and headache.

The arteries are badly filled owing to the distension of the venous system, and consequently the pulse is small and weak. The blood-pressure is low, but may be observed to rise during the act of coughing (Jenner). In the later stages, when the muscular tissue of the heart has undergone degenerative changes, its action often becomes irregular and intermittent.

The veins of the neck are usually distended, and they may pulsate and fill from below. Filling from below is a sign that the valves at the orifice of the jugular veins are incompetent. Forcible pulsation usually indicates that the tricuspid valve is incompetent, but a slight impulse may be the result of the impact of the blood against the tricuspid valve being transmitted through a distended right auricle to the over-filled jugular vein, or it may possibly be due to the systole of the auricle.

An impulse may also be produced in a distended jugular vein by the systolic wave in the underlying carotid artery.

The *physiognomy* of emphysema is characteristic. In the earlier stages of the disease the face is full, the lips are thick, and the mucous surface is congested. At a later stage, when emaciation has occurred, the appearance alters. The lines of the forehead are now deep, the brows knit, the naso-labial folds distinct, the expression careworn. The face is of a faintly bluish tint, the colour being well marked in the lips, which are thickened; the eyes are prominent, and the conjunctivæ injected. At a still later stage there may be well-marked cyanosis of the face. The signs of venous congestion always become more obvious on exertion.

Clubbing of the fingers and toes is often well marked, especially when emaciation has occurred.

The abdomen is usually somewhat distended; the liver and spleen are enlarged from congestion, and assume a lower position than normal; catarrh of the stomach and intestines is apt to cause dyspepsia and flatulent distension. Œdema of the lower extremities is often present in the latest stages of the disease, and dropsy of all the serous cavities with anasarca may occur when there is pronounced failure of the heart. All the symptoms above described become more marked during intercurrent attacks of bronchitis; some, indeed, are present only at such times.

Physical examination; inspection.—The chest tends to undergo enlargement in all its diameters, but particularly in the antero-posterior, owing to exaggeration of the dorsal curve, and to the curvature of the sternum in the opposite direction.

The angulus Ludovici, marking the junction of the manubrium with the body of the sternum, is prominent, and the costal angle is much widened. The vertical measurement is increased by the downward displacement of the diaphragm, and the "oblique diameters" by the ribs becoming more nearly horizontal and the interspaces wider. This form may be modified by the presence of any of the deformities of the chest due to rickets or other causes, to which reference has already been made; but otherwise the general tendency of the chest is to assume a rounded form—the so-called "barrel-shaped chest" of emphysema. The rounded outline is often more marked in the upper part of the chest, whilst in the lower the increase of the transverse diameter is more obvious.

The clavicles are thrown forward, and the sterno-mastoids and other muscles of the neck are tense, giving the neck a short and thick appearance. The supraclavicular hollows may be deep; but if the apices of the lungs are markedly affected the normal depressions here may have disappeared. The curvature of the spine causes the shoulders to be round, and in extreme cases the shoulder-blades may assume almost an horizontal position.

The upper intercostal spaces may present an even surface, but the lower are often depressed. This becomes more marked on inspiration owing to the non-expansion of the emphysematous lung. Bulging of the spaces may be well marked when the patient coughs. The respiratory movements are restricted, and the expiratory act is much prolonged notwithstanding the forcible contraction of the abdominal muscles. The gradual expansion of the chest during inspiration, which is characteristic of health, tends to be replaced by a uniform upward lift, during which the accessory muscles of inspiration stand out in strong relief. In some cases, however, the infra-axillary regions are drawn inwards and the sternum projected forwards, whilst at the same time the epigastric region, instead of bulging during inspiration, may be visibly depressed. This recession of the lower ribs during inspiration is often well marked, and may accompany the deformity of the chest called the "transversely-constricted" thorax, which is usually a relic of infantile rickets. The downward and axial displacement of the heart, combined with the hypertrophy and dilatation of the right ventricle, to which reference has already been made, are jointly the causes of the epigastric impulse commonly observed in emphysema. A horizontal sulcus is observed in some cases to extend across the body from side to side about the level of the lower part of the costal arch. A broad line of dilated venules is often seen in emphysematous subjects tending obliquely upwards on either side along the line of the lower costo-chondral junctions, and across the base of the ensiform cartilage, and therefore corresponding roughly with the attachment of the diaphragm. It is rarely complete posteriorly.

Palpation.—The vocal fremitus is diminished

The impulse in the precordial area is generally feeble owing to the cushion of lung intervening betweeen the heart and the chest wall; but the hypertrophied right ventricle, in the absence of much enlargement of the lung, may cause a heaving impulse in the lower sternal region.

Percussion.—A hyper-resonant note will be found in regions such as the precordial and hepatic, which are normally dull; or dulness may still be present, but over a much diminished area; whilst behind it is by no means uncommon to find well-marked resonance as low as the twelfth rib. Inspiration and expiration make but little change either in the area of resonance or in the pitch of the note on percussion.

Auscultation.—The character of the respiratory murmur varies with the form of the predominant lesion, whether this be of the bullous type or general in its distribution. If "bullous," the breath-sound is weak over the sternum and along the margins of the upper lobes, but harsh beneath the outer half of the clavicle; whilst in the "general" form the breath-sound over the upper lobes is everywhere feeble. It is right to state, however, that the opposite opinion is held by some authors. In place of the normal vesicular murmur audible on inspiration the continuous low-pitched rumbling sound produced by the contraction of the muscles is often very distinct.

When the disease is fully established the expiratory sound is almost invariably prolonged, often very markedly so; in fact, during an intercurrent bronchial catarrh its duration may be so prolonged as to be nearly four times that of inspiration.

These changes in the respiratory sounds are usually most obvious over the upper part of the chest; but when the posterior aspect of the lower lobes is affected the breathing will be weak at the bases, and fine crackling râles may be present there also. These signs are important both as evidence of advanced disease and of œdema of the affected parts of the lung.

At the apex of the heart the sounds are feeble, the characters of the first sound being determined by the relative preponderance of hypertrophy or dilatation of the right ventricle. In the former case it is low-pitched and prolonged, in the latter short and sharp, but weak. The point of maximum intensity of the sounds at the base is lower than normal, and, owing to the increased tension in the pulmonary artery, the second sound is accentuated, and may be reduplicated.

A rough murmur is often audible in cases of emphysema about the sternal end of the sixth left interspace and over the seventh rib, close to the base of the ensiform cartilage. It is systolic in time, usually short, sharp, localised, and superficial, and it often more nearly resembles a rough reduplication of the first sound than a murmur. It may be due to a "white patch" on the anterior surface of the right ventricle, a condition often present in emphysema. The effect of change of position of the body on this sound is variable. It may disappear or remain unchanged. The only importance of the sign arises from the fact that it is very likely to be mistaken for the murmur of mitral regurgitation.

Symptoms of small-lunged emphysema.—In this form of the disease the symptoms are much less pronounced. The most important change in the lungs—the atrophy—is but a part of a general process of wasting in which all the tissues of the body, including the blood, share alike. The respiratory needs are therefore less, and they may be adequately met by a smaller pulmonary area. The capacity for exertion is limited because of the feebleness of muscular power; and, in the absence of effort, there may be little or no dyspnœa.

Another point of difference from the variety just considered is that atrophic emphysema is rarely complicated by attacks of bronchial asthma; but intercurrent bronchitis may induce dyspnœa which, although differing in its mode of onset, is hardly less in degree than that which characterises the asthmatic paroxysm.

Physical examination; inspection.—The emaciated and withered appearance of the subjects of this form of the disease has already been mentioned. The evidences of venous obstruction, such as cyanosis and clubbing of the fingers, are absent; as also are the effects which that condition produces in the size, shape, and position of the heart. The chest assumes the barrel shape as a result, not of a process of enlargement, but of "shrinkage" in all its diameters, and especially in the lateral. The gradual diminution in the size of the lungs is necessarily accompanied by a recession of the ribs, which assume a more oblique position. The interspaces from the first to the fourth on the front of the chest are often both wide and deep; but the increased obliquity of the lower ribs tends to approximate them, so that the interspaces may be obliterated, or adjacent ribs may even overlap each other.

Inspiration is shallow, the rigid thorax moves as a whole, the upper interspaces recede, and descent of the diaphragm is restricted.

Percussion.—The note is hyper-resonant, but it tends to be clearer in tone and more tympanitic in quality than in the large-lunged variety. The area of precordial dulness is not diminished and may possibly be increased. The former statement applies also to the hepatic dulness.

Auscultation.—The breath-sound is weak, but the expiratory sound is not prolonged to nearly the same extent as in large-lunged emphysema. Adventitious sounds are not necessarily present, but the coexistence of chronic bronchitis is so common as to make their complete absence very rare; fine and medium bubbling râles may be heard over the bases of both lungs. Fine crackling râles may be audible over the same area if œdema is present.

Other pulmonary complications will give rise to the auscultatory signs by which they are usually characterised, modified to some extent by the presence of emphysema.

Symptoms of localised emphysema.—On reference to what has been stated as to the mode of production and common sites of this variety of emphysema, it will be seen that the symptoms must necessarily depend upon the condition to which it is secondary. It may, however, be repeated that it is frequently a sequence of tuberculosis, and its presence at the apex of a lung should suggest the possibility of such a connection.

An enlargement of one lung or of a portion of it, consequent on disease and contraction of the opposite lung, is not necessarily due to emphysema; it may be a true hypertrophy. The test by which the two conditions are distinguished is that of functional activity. If this is increased, the enlargement must be regarded as hypertrophy; if diminished, as probably due to emphysema: in the former case the breathing is puerile, in the latter it is usually feeble with prolonged expiration.

Symptoms of acute vesicular emphysema.—As already stated this condition is only recognised as a form of the disease in deference to tradition.

It originates during a state of extreme dyspnœa, the urgency of which it doubtless increases; but the result to the patient is probably determined almost invariably by the nature of the exciting cause and not by the effect produced upon the lungs. Cyanosis may very likely be observed during the attack.

The chest will be in a condition of extreme inspiratory distension. The nature of the breath-sounds and adventitious sounds will vary with the exciting cause.

The **diagnosis** of the large-lunged form of emphysema rarely presents much difficulty. It is suggested by a history in which cough and dyspnœa are prominent features, or by the patient being engaged in some occupation known to involve severe muscular effort; it is confirmed on examination by the alteration in the form of the chest, the hyper-resonance on percussion, diminished movement and feeble respiratory sounds—signs which are present on both sides of the chest.

Error has apparently arisen at times from pneumothorax being mistaken for this form of emphysema. In such cases the methodical examination of the chest has probably been neglected, and undue reliance placed upon one step in the process, possibly on percussion. In pneumothorax the enlargement of the affected side, the obliteration of the interspaces, the absence of movement contrasting with the increased movement of the healthy side—if it be healthy, the displacement of the heart to the sound side, the more amphoric note on percussion, and the absence of the breath-sounds, or their amphoric quality, are signs which combine to form a picture that, in well-marked cases, should be unmistakable.

It is possible, however, for a collection of air, confined by firm adhesions to a very small part only of the pleural cavity, to give rise to signs which may be mistaken for those of emphysema. Such a case, due to the rupture of an emphysematous bulla near the base of the lung, came under my own notice. It is sufficient to mention it as a possibility to be borne in mind without discussing in detail the diagnosis of a condition of such rare occurrence.

Aneurysm of the transverse part of the arch of the aorta compressing the trachea may be mistaken for emphysema with bronchitis. The tracheal stridor and brassy cough, the dulness, or at any rate the absence of increased resonance over the manubrium, and the loud tracheal breathing over the same area usually suffice to prevent error.

"Emphysema and bronchitis" is occasionally the diagnosis on admission to hospital of cases in which the primary disease is really stenosis of the mitral orifice; cardiac failure, pulmonary engorgement and œdema have supervened, and the murmur has disappeared. After a few days of rest and treatment considerable improvement as a rule takes place, the murmur again becomes audible, and the true nature of the case is then obvious.

True cardiac dyspnœa is distinguished from that accompanying emphysema by its "panting" character; but failure of the right heart often follows upon long-standing emphysema, and the dyspnœa is then the resultant of the two conditions and partakes of the characters of both.

An examination of the sputum for tubercle bacilli should always be made in cases of emphysema and bronchitis, particularly in such as are accompanied by marked emaciation. In the fibroid form of pulmonary tuberculosis, which is often associated with emphysema (not so-called "fibroid phthisis"), bacilli may be absent and the true nature of the disease may only be discovered on autopsy. The absence of pyrexia in such cases is not a distinguishing symptom of much value; fibroid tuberculosis being often unaccompanied by fever, at any rate for intervals of considerable duration.

The diagnosis of the atrophic form of emphysema is but rarely attended with difficulty.

Prognosis.—True emphysema, that is, dilatation with atrophy, is a permanent condition, with a decided tendency to advance. But whether it increase, and if so, at what rate, depends chiefly upon the continuance of the exciting cause, which, in the great majority of cases, is the cough of catarrh or bronchitis. If the patient is able, by change of residence or in other ways, to shield himself from adverse conditions of climate, the disease may remain stationary. Under any circumstances its course is chronic, and life only becomes endangered when complications arise.

The extent of the lesions will naturally influence the prognosis; but the effect produced upon the heart and circulation is a far more important factor in determining the probable duration of life. As dyspnœa is the chief evidence of this effect, its degree during rest and on exertion becomes one of the main elements in prognosis. The condition of the veins of the neck as to over-distension, pulsation, and filling from below, is an important guide to the state of the right side of the heart.

The existence of enlargement of the liver, œdema of the legs, ascites, and albuminuria marks an advanced stage of cardiac failure.

The presence of renal complications, particularly chronic interstitial nephritis, is of especial importance in prognosis.

Treatment.—Sufferers from emphysema rarely ask for advice on this ground alone, the disease being one of the existence of which the laity may be said to be ignorant. As a rule, no complaint is made of the accompanying dyspnœa; the patient has become so habituated to it that he has ceased to regard it. In the majority of cases the condition is discovered when an intercurrent attack of bronchitis leads to an examination of the chest. Atrophy of the alveolar walls, destruction of the capillaries, and wasting of the elastic tissues are changes which cannot be repaired; and a return to the normal state is only possible in the cases of temporary over-distension which occur for the most part in young subjects, as a result either of laryngeal obstruction, spasm, or whooping-cough, or of bronchitis accompanying an acute disease, such as measles.

Much, however, may be done to stay the progress of the disease by shielding the patient from further attacks of bronchitis, or by advising a cessation of any occupation which necessarily involves a strain upon the respiratory organs. Treatment may also be usefully directed towards the relief of the secondary effects upon the heart and circulation.

Emphysema once established undoubtedly disposes to bronchitis; it is therefore of the first importance that all known causes of catarrhal inflammation should be carefully avoided. Those whose means permit will be well advised to spend the winter and spring in a warmer climate than is to be found in this country at such times; many sufferers, however, although they know this full well, are prone to delay their departure unduly, and an early November fog finds them still here; the result too often is a severe attack of bronchitis and much increase in the emphysema. Persons who are unable to leave home, if they hope to escape an attack of bronchitis, must exercise the greatest care in avoiding cold north and east winds, foggy and damp air, over-fatigue, or sitting in draughty rooms, and anything likely to give rise to a chill. Notwithstanding its unsightly appearance, a respirator, or woollen "comforter" covering the mouth, by warming the incoming air is of real service in warding off attacks of bronchial catarrh.

The conditions which give rise to increased pressure within the air-passages have already been described; it will be sufficient, therefore, to state that it is absolutely necessary for the sufferer from emphysema to avoid them if he wishes to escape an increase of his disease.

The effect upon the respiration is a useful test as to whether any form of exercise is harmful either in kind or degree; if it causes dyspnœa it should be avoided. The bowels should not be allowed to become confined, as, in addition to the gastro-intestinal derangements likely to ensue, much harm may be done by straining efforts in defæcation.

In the article on "Aerotherapeutics" (vol. i. p. 315) a full description is given of the various forms of apparatus used in the application of condensed air to the body as a whole, and of condensed or rarefied air to the respiratory surface in emphysema. Notwithstanding that much has been done in recent years to render our knowledge of this branch of treatment more exact it is still but little used in this country. This is doubtless due to the fact that patients are rarely under treatment for emphysema apart from its complications; and also to the small number of compressed air baths available for use.

The condition of the lungs in emphysema indicates that expiration into rarefied air should afford relief. This proceeding causes a diminution in the amount of residual air, and an increase in the volume of inspired air; thus a partial retraction of the lungs and a rise in the position of the diaphragm are brought about. These changes are accompanied by a lessened circumference of the chest, and by an increase in the vital capacity and of the force of inspiration and expiration. The apparatus of Waldenburg, of which a description will be found in the article on "Aerotherapeutics" (*loc. cit.*), is most suited for this form of treatment. Expiration into rarefied air produces a sense of extreme constriction within the chest and certainly diminishes the amount of residual air. The "vital capacity" of patients with emphysema under treatment by this method undoubtedly increases; but this result cannot be accepted as an absolute proof of its value, as it also follows the use of the apparatus by those whose lungs are structurally sound, practice enabling the individual to obtain a better result.

The results obtained from expiration into rarefied air are, however, much less satisfactory than those which attend the use of compressed air applied to the body as a whole.

I have given a prolonged trial at the Brompton Hospital to the use of the compressed air bath in the treatment of emphysema associated with bronchitis, and am able to support the favourable opinions expressed by Dr. C. Theodore Williams and others as to its great value.

Patients almost invariably state that they breathe more freely whilst in the bath and after a considerable number of baths (from 20 to 30 or more) have been taken; this feeling becomes continuous, and has remained whilst the patients have been under treatment. The greater capacity for exertion which follows the use of compressed air baths in emphysema has been tested by observation of the gradually increasing facility with which patients thus treated have been able to mount a flight of steps which leads from the basement, where the bath is situated, to the "gallery" (wards) occupied by them. Patients who at first were obliged to use the lift to return to their ward, or were only able to climb the stairs with many halts to take breath, have been enabled gradually to reduce the number of stoppages on the ascent; and many have at length been able to return from the basement to the uppermost floor without stopping once.

In addition to the greater freedom of respiration and increased capacity for exertion, the cough becomes less frequent and the quantity of expectoration is reduced.

It is not quite clear how these favourable results are produced. In a healthy person the effect on the respiratory organs of submitting the body as a whole to air gradually condensed to the extent of three-sevenths or one-half an atmosphere is to cause diminished frequency of respiration, enlargement of the lungs, increase of the vital capacity, and probably also an increase in the amount of oxygen absorbed. The change is attributed to the greater density of the air, and consequently to the increased amount of oxygen supplied to the lungs. The respiratory power and the elasticity of the lungs, both during and after the bath, are increased; the chest is enlarged in all its measurements, and the diaphragm assumes a lower level. In the subjects of emphysema, however, the effect of the bath is to cause a reduction in the size of the chest, as ascertained by measurement of the circumference; and also in the amount of distension of the lungs, as proved by the reappearance of dulness in the precordial and hepatic regions. The diaphragm is raised instead of being lowered, and epigastric pulsation may be replaced by an impulse more nearly in the normal situation of the apex beat of the heart.

It appears probable that the condensed air penetrates into parts of the lungs which have been long unused in respiration, and in which air has been, so to speak, imprisoned at a high pressure; the escape of this air is facilitated and contraction of the lung follows.

In some cases the improvement following the use of the bath is but temporary, and in cases of emphysema accompanied by asthma I have observed very severe attacks of dyspnœa to follow very shortly after a bath. If this should occur after the second bath, it is generally better to discontinue its use. Many cases of asthma are, however, greatly benefited by this method of treatment.

For the details of this method the reader is referred to the article on "Artificial Aerotherapeutics," vol. i. p. 310.

The treatment of an attack of bronchitis occurring in a patient the subject of emphysema is not materially modified by the latter complication; but the duration of the attack is sensibly prolonged, and the danger to life is much greater, owing to the loss of power of expectoration which results from the diminished elasticity of the lungs.

Spasmodic dyspnœa often accompanies an attack of bronchitis, and requires the use of such remedies as stramonium, lobelia, belladonna, grindelia, or iodide of potassium in large doses, in addition to the ordinary drugs used in the treatment of bronchitis. The desirability of employing morphia in such cases will depend chiefly on the relative preponderance of the spasmodic or the catarrhal factor. The nearer the attack approaches in character to one of true asthma the greater is the probability of relief from a subcutaneous injection of morphia; whilst, on the other hand, if the dyspnœa be chiefly due to the accompanying bronchitis, the use of morphia may be attended with the greatest danger. The history of previous attacks, the mode of onset, the presence of pyrexia, the character of the adventitious sounds—for instance, the presence of fine or medium bubbling râles, indicative of an affection of the smallest bronchi or of the alveoli—and particularly the condition of the bases of the lungs, are some of the points to be considered in determining such a question. In the treatment of the attacks of wheezing, so often met with in emphysema, apart from any serious bronchial attack, a stimulating liniment containing turpentine and iodine rubbed into the chest is often of much service. Iodide of potassium in doses of five, eight, or ten grains three times daily, in combination with extract of stramonium and carbonate of ammonia, generally affords relief. In the intervals of comparative freedom from such attacks, and often throughout the winter months, the administration of cod-liver oil is hardly of less service than in cases of pulmonary tuberculosis. It is of special benefit when nutrition is failing, as is commonly the case in advanced stages of the disease, and in the atrophous emphysema of the aged. Iron in combination with spirits of chloroform is often taken by patients with emphysema with much benefit.

Turpentine, terebene, and balsamic remedies are of service where expectoration is excessive; this symptom is, however, due to the accompanying bronchitis, and its treatment is described in the article on that subject.

Cyanosis is an indication for venesection, and the necessity is urgent when there is evidence of great over-distension of the right side of the heart, with tricuspid regurgitation, pulsation in the jugular veins, and œdema of the feet. Digitalis should be given as soon as the blood has been drawn; and its use may be necessary in cases which are not so advanced as to require venesection.

When, as is not uncommonly the case, emphysema supervenes on bronchitis of gouty origin, the existence of this factor in the case must not be overlooked in the treatment. The same statement applies to the coexistence of chronic interstitial nephritis. It must not be assumed at once that the presence of a small quantity of albumin in the urine is due merely to renal congestion; search should be made for casts.

It is of great importance in cases of emphysema accompanied by attacks of dyspnœa, occurring at night, that the patient should not take a heavy meal at seven or half-past and retire early to bed; by so doing he is very likely to induce an attack. Full time should be given for digestion, and the lighter the evening meal the better; such patients should dine in the middle of the day.

Few conditions apart from bronchial catarrh are so likely to induce an attack of dyspnœa as flatulent distension of the stomach. This is chiefly to be avoided by attention to diet; and these patients are nearly always well aware what food suits them and what does not. A mixture containing bicarbonate of soda, tincture of nux vomica, compound tincture of cardamoms or tincture of ginger, with a bitter infusion, taken half an hour before meals, may prevent such an attack. A dose of blue pill, taken twice a week at bedtime and followed in the morning by a saline purge, is often beneficial in middle-aged subjects of the disease who are well nourished and have a tendency to gout.

INTERLOBULAR OR INTERSTITIAL EMPHYSEMA

The escape of air into the connective tissue of the lung produces a condition to which the above name is applied.

As stated in the previous section, it has nothing in common with emphysema of the lungs but the name.

The air appears as rows of beads beneath the pleura and in the substance of the lung.

Wounds of the lung or rupture of the air-vesicles from over-strain during violent cough are the most common causes of the affection.

I have specially observed it in connection with laryngeal diphtheria, generally after tracheotomy had been performed; but it may occur independently of that operation. The air, as pointed out by Dr. Champneys, passes from the tracheotomy wound downwards into the thorax behind the deep cervical fascia. From the mediastinum it may spread along the connective tissue surrounding the bronchi and vessels, and may appear on the surface of the lung as small beads of air beneath the pleura.

Mediastinal and interlobular emphysema may occur in diphtheria when tracheotomy has not been performed, probably from rupture of vesicles upon the surface of the lung; and pneumothorax, from perforation of the pleura, may follow.

Pathology.—The following extracts from the post-mortem register of the Middlesex Hospital (2) illustrate the changes met with in cases of interstitial and mediastinal emphysema:—

Case 1.—Male, age 3½ years. Diphtheria; tracheotomy. Extreme sub cutaneous emphysema of the face, neck, and trunk; collapse of both lungs; mediastinal and subpleural emphysema.

Case 2.—Female, æt. 5. Diphtheria; tracheotomy. Lungs fully distended; no collapse; air in anterior mediastinum; membrane on fauces and in larynx, trachea, and bronchi.

Case 3.—Female, æt. 5. Diphtheria; tracheotomy not performed. Emphysema of root of neck; mediastinal, interlobar, and interlobular emphysema; pneumothorax (R); pulmonary collapse.

Case 4.—Male, æt. 5. Diphtheria; tracheotomy. General emphysema of subcutaneous cellular tissue of neck, trunk, and arms; lungs almost completely collapsed from double pneumothorax; air in mediastina and around roots of lungs; membrane on tonsils and in larynx, trachea, and large bronchi.

Case 5.—Male, age 2 years. Diphtheria; tracheotomy. Larynx completely blocked with membrane, which extended throughout the trachea and main bronchi; lungs collapsed in patches; emphysema of anterior mediastinum.

Case 6.—Male, age 11 years. Diphtheria; tracheotomy. General emphysema; membrane in trachea and bronchi of left lung, latter collapsed; marked emphysema of anterior mediastinum.

Case 7.—Female, age 4 years. Diphtheria; tracheotomy. Interlobar emphysema on right side; air in anterior mediastinum; membrane as far as secondary divisions of bronchi; numerous areas of pulmonary collapse.

The preceding cases illustrate the lesions commonly found in association with interlobular emphysema when that condition occurs in diphtheria; the most important being general emphysema, pneumothorax, and pulmonary collapse.

Symptoms.—In all the cases above described in which tracheotomy was performed there would necessarily be urgent dyspnœa at the time the trachea was opened. The dyspnœa would then be relieved, but the occurrence of mediastinal and interstitial emphysema is accompanied by an increase in the dyspnœa. If pneumothorax supervenes, the dyspnœa becomes extreme.

Double pneumothorax is necessarily quickly followed by death.

The breath-sounds would almost certainly be weak or absent if the connective tissue of the lung were infiltrated with air. Pneumothorax would be characterised by its ordinary physical signs.

Interlobular emphysema is rarely recognised during life. It may be suspected when subcutaneous emphysema is present, or when pneumothorax occurs. The latter is a serious complication. It is probable that the condition here described is often present but is unsuspected, and that the air is absorbed when recovery takes place.

No definite *treatment* can be adopted for the condition.

J. K. FOWLER.

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J. K. F.

ON ASTHMA AND HAY FEVER

ASTHMA is a paroxysmal dyspnœa which often manifests itself quite suddenly and from a great variety of causes; and which may subside again with like rapidity. The respiration in the intervals may or may not be normal.

It is usually divided into primary or idiopathic and secondary or spasmodic asthma. The latter kind appears to originate from more or less bronchial catarrh. In the management and treatment of the affection it will be necessary to take this distinction fully into consideration; but seeing that spasmodic asthma can, and certainly does, occur independently of local and chronic irritation, I shall first consider it in its simplest form as the primary disease. I shall first describe the features of an attack of asthma, and then discuss in natural sequence its causation, its pathology, and the general management of the asthmatic patient.

Hay fever is often a spasmodic asthma in its purest form, so that the two maladies will be considered in common.

Symptoms.—*The asthmatic paroxysm* may come on at any time. A susceptible or morbidly paroxysmal subject—to be paroxysmal more or less is a universal attribute of organic action—comes into contact with, say, some animal or vegetable exhalation; eats some indigestible article of diet, or something that, while innocuous to the mass of mankind, is known to be in some way prejudicial to particular individuals; or in some other of many ways taxes his range of accommodation beyond the margin of its power, and within a few minutes an attack will begin. The beginning is said to be most frequent during the night, when the patient has had his first sleep; for instance, at two or three in the morning he suddenly awakes with a stuffy feeling in his chest, and within a short time he is in the throes of an attack of asthma. Thereupon he is compelled to sit up in bed, perhaps to rush to the window; the head is fixed, the shoulders raised, the hands are often planted well down upon anything firm upon each side to give purchase to the respiratory muscles, and so the sufferer sits labouring at his breath. Sometimes he bends forward, sometimes stands leaning upon some support; but the object in all cases is the same, to give the respiratory muscles better or more fixed support from which to act.

The pulse is usually but little affected, and the temperature is normal.

In bad cases the face is of an ashy pallor, or it is gray and leaden, or dusky from want of oxygenation of the blood; the skin is covered with perspiration, the eyes may look prominent, the nostrils may be dilated. Few diseases produce appearances so distressing and so grave, and yet it can fortunately be said that an attack of spasmodic asthma never kills. Probably it supplies its own corrective in this, that after a certain time of agony, or certainly of intense distress and anxiety to the patient, the irritated centres become exhausted, the spasm is gradually relaxed, and the patient sinks to sleep. In some cases the relief appears to be absolute; in the majority, however, it is only comparative, and more or less oppression is experienced for a day or two, sometimes for many days; or the malady may abide with the patient more or less continuously.

During an attack, although the patient is making violent efforts with all the respiratory muscles, the actual movements of the chest wall are little indeed. The chest may plunge, but there is no expansion of the thoracic cavity. On the contrary, as the chest walls are pulled outwards all the more yielding parts are depressed and thus the intercostal spaces become troughs. The epigastrium may be hollow or full; the suprasternal and supraclavicular spaces are greatly exaggerated.

The actual condition of the chest during a paroxysm has been the subject of some discussion. The generally accepted description, following Salter, is that it is in a state of over-distension, the diaphragm being depressed and the upper part of the abdomen being full (Hyde Salter and Biermer). The movement is much restricted, and thus there is a very short, abortive, suddenly pulled-up inspiration, and an expiration perhaps four or five times as long as it should be. The percussion of such a chest gives a hyper-resonant note. But I am sure that I have seen, as stated by Riegel and others, another form of chest, where the lower parts, if not retracted as some contend, were not unduly distended, and where dulness rather than hyper-resonance was detected. And I have always supposed that in these cases the obstruction in the smaller bronchia is so extreme as to lead to a state of collapse. Wilson Fox offered this explanation, and the whole subject will be found discussed in his posthumously published work, *A Treatise on Diseases of the Lungs and Pleura.*

The attack is, however, by no means always sudden in its onset, perhaps not generally so. More or less wheeziness and constriction of the chest may exist for a day or two beforehand; there may be a short, rather hollow cough, and, if the dyspnœa be at all pronounced, much weariness on exertion. Thus a mild or threatening attack may be recognised by the onlooker in the disinclination to all movement generally shown by persons thus affected.

If the chest of the asthmatic be auscultated during the paroxysm the chief feature is an almost complete absence of respiratory murmur. The chest, as I have said, plunges, but there is no corresponding inspiratory wave; there are sibilant rhonchi and muscular rumbles, and a variety of odd noises, but no real ingress of air; and with the expiration there may be, perhaps, a long, distant, soft sibilus, the sole evidence of the respiratory ebb. The disease is often unequally distributed: one side, or this or that portion of one lung is affected more than the other; the asthmatic storm flits about the lung, now here, now there, and when the disease is thus unilateral or partial it is liable to repeat itself thus; so that we surmise that there is some local disease in the form of bronchitis, emphysema, adherent pleura, and so on, which exercises a permanently determining effect.

It is said that when an attack is over the patient is free from liability to a recurrence for some time. But in all probability this depends upon the past duration of the asthmatic habit. Asthma, like gout, although in its earlier years markedly periodic in recurrence, tends, as the patient grows older, to become erratic in its manifestations, both as regards the time of its appearance and the form in which it comes. So that whereas in its earliest appearances it comes and goes, maybe with some special regularity, later in life it comes but it does not go, and the patient thenceforward is subject to a more or less chronic bronchitis. And in all old cases, in which the chest is damaged by the repetition of the paroxysms, emphysema is produced with its attendant chronic bronchitis.

As the attack ends, expectoration usually begins. In the earlier stages of the paroxysm the bronchial tubes—to judge from the character of the signs—are dry; the prevailing sounds in the chest being wheezing sibilus and rhonchus. Under the intense oppression the patient longs to expectorate, but is not able to do so. But when subsidence approaches, small gray pellets of mucus, of characteristic appearance, "like tapioca" (Salter), "often filamentous in shape like boiled macaroni" (8), begin to appear and gradually increase. In association with the appearance of crepitation in the chest the sputum becomes more and more copious, thin and frothy, till it may reach some considerable quantity. Blood seldom comes, but in severe attacks it may, and, if so, generally in streaks.

The clinical history of spasmodic asthma is, however, by no means completed by this description of a characteristic attack. There are several other irregular states that to my mind are no less parts of the disease. First of all there is hay fever, when brow ague, coryza, a more or less general disagreeable stuffiness of the respiratory tract, rendering nasal breathing a difficulty, and producing a more or less chronic wheeziness and distress, last throughout the summer months. At times there is some slight febrile reaction, but it is not often great in degree; there is rather the subjective feeling of lassitude and heat than the objective evidence of actual pyrexia; and perhaps, on the whole, these rather indefinite symptoms are the more usual mode of its attack, although a definite attack of spasmodic asthma is by no means uncommon.

Paroxysmal sneezing is another way in which the asthmatic respiratory tract explodes. If we study asthma or the history of the asthmatic in any comprehensive way, we cannot but be sure that, either as a substitute or as part and parcel of the asthmatic attack, this sneezing must be taken into account. It is often found in asthmatic subjects and in asthmatic families, in which one member may have asthma and another exhausting paroxysms of sneezing; moreover, it often goes with asthma, the sneezing gives the impetus of origin to the asthma, the irritation in the upper air-passages gradually spreads down the bronchial tubes, and asthma more or less severe results. In the history of asthma cases of this sort are to be found in numbers; but they need not be more particularly described, for they are fully dealt with under an appropriate heading. I may say this, however, that most of the cases of sneezing I have met with have been in women, which is against the rule that prevails in asthma, in which case men are in the proportion of two to one. There are authors who attribute such cases largely to local disease in the nose, and believe that they are to be relieved, as also the asthma that accompanies them, by local treatment of the nasal mucous membrane. One case may be quoted that illustrates several of these points: a man, aged twenty-eight years, who came of healthy, non-asthmatic stock, fell off a bicycle and smashed his nose. Ever since that time, now twelve years ago, he has required for his daily use six or seven pocket-handkerchiefs, and now he has become asthmatic. His asthmatic attacks come on every month or six weeks, and last from half an hour to a day and a half. He has had his nose treated with decided benefit to his asthma, but he derives most benefit from smoking medicated cigarettes. With reference to the nose the experiments of Lazarus (7) are of much interest. This observer has demonstrated a certain relationship between the nasal mucous membrane and the bronchial muscles, so that, by the application of weak electrical currents to the nasal mucous membrane, he was able to register a distinct increase in the intra-bronchial pressure.

There is at least one other affection that I would include in the clinical history of asthma, namely, the paroxysmal bronchitis of infants and young children. It has always seemed to me that one of the most interesting features in the study of medicine is the modification that disease undergoes in the successive periods of life. It is not certain, perhaps, that disease is so modified, but there is plenty of evidence to point in that direction. For instance, when a man who in earlier years had acute rheumatism is attacked in middle age with well-marked gout, we may suppose that a common factor has been modified, so that what did produce acute rheumatism at a later date produces gout. Now, as regards asthma, I believe that something of this kind takes place. Asthma, as I shall presently show, is largely a disease of childhood, but it is not clearly present in the earliest years. Hyde Salter has seen two cases in infants of fourteen and twenty-eight days, but such instances are very rare. It frequently begins to appear at six, seven, or eight years of age, and there are a fair number of suspicious cases at earlier periods than this. In infancy, if asthma exists, as I believe it does, it shows itself as a bronchitis, so far as the physical signs go; but, if so, it is a strange and interesting bronchitis, apart from the physical signs. It comes on with remarkable suddenness; it is mostly associated with fever; it is generally attributed to chill by the relatives: but there are reasons for thinking that it owns a much greater variety of causes, such as over-excitement, errors in diet, dentition, and so on. It clears up with remarkable celerity and certainty; it often leaves the child no worse than it was before the attack. Such attacks as these occur in a particular class of children,—children that give conspicuous evidence either of coming of nervous stock, or of nervousness and excitability in themselves. The whole history of these cases is explosive and nervous; and it may well be that, in the early history of the child's life, the ribs and other parts of the respiratory apparatus are not sufficiently developed to produce asthma, as we expect to see it; so that the mode of the disease is atelectatic or bronchitic. Asthma, in its ordinary manifestations, requires certain conditions of respiratory power, which, in all probability, the thoracic walls do not readily supply at that early period. Moreover, there can be no doubt that in the seven stages of our existence—and this answers to some extent the question I have already mooted—our various viscera change places in their relative importance, not only in their several bearings upon the well-being of the organism, but also in the absolute degrees of their activities; now one, now another, becoming a centre of excitement and explosion, and thus of break-down in ill-balanced organisations. In infant life the stomach tends to play the part of the spoilt darling, and the lungs often have to pay the penalty for its caprices. However well it may be, it would appear that often, as with many another ill-bred person suddenly thrown into a position of trust and responsibility, it is not equal to the occasion; the household's teeth are set on edge, and pulmonary catarrh or œdema or collapse is set up. It seems to me that these sudden storms, which so expend themselves on the lungs, or in the achievement of pyrexia, have much similarity to asthma in their sudden mode of outburst; they involve a similar area, and may therefore not inappropriately be considered in the youngest children as the representative of asthma. And having diverged from the immediate subject to introduce that of the correlatives, substitutes, or derivatives of asthma, I shall briefly indicate several other diseases that may in this respect be considered with the gastro-pulmonary fever that I have just mentioned. Some persons, for instance, have laid stress upon psoriasis in this connection. I have myself known of a case where asthma and psoriasis seemed to alternate in the same person, and I have also come across this curiously suggestive alternation as regards eczema. The mother of a family is the subject of spasmodic asthma. She has had four children. In the first and third pregnancies she had no asthma, and in each child bad eczema appeared, and death resulted in one from convulsions. In the second and fourth pregnancies the mother had bad asthma, and the children hitherto have been healthy. I find from a collation of my notes that no less than seven out of 125 cases of asthma were associated with severe eczema, and in two or three of these as the eczema went the asthma came. Carl v. Noorden is perhaps the most recent author who has drawn attention to the frequency with which asthma is associated with eczema, but the connection has long been noted.

Again, I have elsewhere thrown out the suggestion that some of the cases of paroxysmal sneezing, which, as I have said, are undoubtedly part of the complete picture of asthma, may also be a part of the history of Raynaud's disease; for they go with weak peripheral circulation, with waxy fingers, with chilblains, and so on. All three are probably due to allied causes; and although in all the three the results are of different order (in the case of the nose, turgidity; in that of the extremities, cadaveric blanching or chilblains; in the case of the lung, a supposed spasm of the muscles of the smaller bronchi, leading to a temporary collapse of the affected part of the lung), yet the clinical history in all of these is not unlike. In all there is the same tendency to suddenness of onset, the same sort of rhythmical association between flux and its opposite, the same curious vagaries of onset from causes that seem quite inadequate.

Of other affections that surely belong to the same category is that form of looseness of bowels which is so common in nervous subjects, and in excitable children, where the mere ingestion of food seems sufficient to provoke a stomach-ache and a profuse liquid evacuation from the bowels. This is perhaps the very commonest of the kind. Another is urticaria, and it is not uninteresting to note that it is sometimes associated with or replaces asthma, as a case of asthma produced by contact with cats will show. I have records of three such cases.

To complete the clinical picture, it must be said that although a certain number of cases are inexplicable explosions, and all of them own something of that character, yet many, perhaps most, have a local exciting cause—a cause inefficient, it may be, to produce any such disagreeable effects under healthy conditions of the nervous centres, but which under diseased or ill-regulated conditions becomes an active source of worry and excitement. Such things are pneumonia, bronchial catarrh, whooping-cough, and so on. Eighteen out of 125 are attributed to such a cause.

Causation.—As to *sex*, it is usually stated that asthma occurs twice as often in men as in women: 73 to 50 in my own cases. It might have been anticipated that the less stable centres of the woman would be the more likely to show a predominance, but it may well be that the instability of womanhood works off in other ways. Salter considers that this unexpected incidence of a nervous affection upon males may be an argument in favour of the existence of some organic change in the lung.

Of *age*, Hyde Salter remarks that it is a commonly received opinion that asthma is a disease of advanced life, but that it is not confined to any one time of life; so far, indeed, is it from being peculiarly a disease of the old, that a larger number of cases take origin in the first ten years of life than in any subsequent decade. This seems to me quite a correct statement of the matter, for I find that, of cases in which the point is noted, 50 began in children of ten years and under, 31 in males, 19 in females: it is interesting to note that the youngest case was in a little boy 3½ years old, whose father suffered from hay fever and asthma, and who was said to have been quite cured by local treatment of his nose with the cautery.

In 23 cases the disease began between ten and twenty, 13 being males and 10 females. In the period from twenty to thirty only 12 cases are reported, 4 males and 8 females. In 36 cases, 24 men and 12 women, the disease arose after the age of thirty. These figures indicate, too, that the excessive incidence of the disease upon males is all along the line, with the exception of the decade from twenty to thirty, in which perhaps the numbers are not sufficient to base any conclusions upon. Thus, in seventy-three cases out of 121 asthma began in subjects under full age.

Hyde Salter's table of the age at which people have become asthmatic is as under:—

During first year . .	11 cases	31 per cent
From 1 to 10 . . .	60 ,,	
,, 10 ,, 20 . . .	30 ,,	12·8 ,,
,, 20 ,, 30 . . .	39 ,,	17 ,,
,, 30 ,, 40 . . .	44 ,,	19 ,,
,, 40 ,, 50 . . .	24 ,,	9 ,,
,, 50 ,, 60 . . .	12 ,,	5 ,,
,, 60 ,, 70 . . .	4 ,,	1·7 ,,
,, 70 ,, 80 . . .	1 ,,	0·4 ,,
	225	

Heredity.—Of 123 cases, 50 showed a well-marked neurotic inheritance of one form or another; in 25 it was the direct transmission of asthma or of hay fever. In 8 more one or other of the parents had had rheumatic fever; in other families there is a history of megrim; in others somnambulism and diabetes existed. And, indeed, in all this group of diseases—in asthma, hay fever, and paroxysmal sneezing—the number of the nervous phenomena that are to be found in the different members of the family is conspicuous. Berkart, in some carefully selected cases, found that in 16 per cent one or other parent suffered from asthma.

Of other remoter causes one must certainly mention an idiosyncrasy on the part of the subject—"individual constitution," as Wilson Fox calls it. What this is we know no more than why certain foods, which for the majority of mankind are perfectly harmless, for a small minority are active poisons. For my own part I am inclined to doubt whether this constitution is ever wanting in the case of asthma, even though diseased conditions be actually present that seem so immediately provocative of an attack as naturally to be regarded as sufficient causes.

Immediate causes.—Of the two groups of cases into which we divided asthma at the outset, perhaps almost enough has been said incidentally about idiopathic asthma. Given a certain morbid sensitiveness of the nervous centres, anything seems capable of producing an attack. It may be a nervous shock, over-fatigue of mind or body, too monotonous a habit of living, too little exercise, too much food, indiscretions of one kind or another in diet, changes of temperature, changes of climate, a thunder-storm—changes in the weather seem to be particularly prone to induce asthma, microbic organisms in the atmosphere, or emanations of various kinds from animate (cat or horse asthma), inanimate bodies (hay), and so on.

Of some of these emanations one would not wonder that dust, fog, or pungent fumes of various kinds should now and again be responsible for the production of the disease. But the peculiarity of asthma would seem to be that it is evoked by irritants that under ordinary circumstances are no irritants at all (Salter). "One asthmatic is obliged to expatriate himself in the hay season and take a sea-voyage; another cannot stay in a room in which a bottle of ipecacuan is opened; a third cannot stroke or nurse a cat; another cannot go near a rabbit hutch; another is immediately rendered asthmatic by the neighbourhood of a privet hedge; another cannot sleep upon a pillow stuffed with feathers; another cannot use mustard in any shape, or bear it near her, so that she dare not even apply a mustard plaster; and one young lady I know who dare not pass a poulterer's shop."

I have myself knowledge of two cases of cat asthma. In one of them the existence of cats is the bane of life, for before accepting an invitation she is obliged first to ask, "Is there a cat?" An attack of urticaria and coryza followed by asthma has been noticed to come on within ten minutes of having stroked a cat. At other times sitting in a room in which there was a cat, without any actual contact with it, was sufficient to produce a bad attack, beginning within ten minutes of entering the room.

Professor Clifford Allbutt tells me of a little boy in whom horses work similar effects. He cannot, therefore, ride in carriages or cabs; and it has been necessary to let him run home and get wet through, rather than incur the greater evil of asthma, likely to be provoked by a ride in a cab with his mother.

Such statements as these, Salter truly says, one would hardly believe, were not their reality placed beyond doubt; there is neither invention, nor imagination, nor exaggeration about them.

Surgeon-Major Lethbridge Swayne, practising in Aurungabad, tells me that asthma is quite common there in association with malaria; and that asthma often ushers in an attack of malarial fever, and has done so in his own case several times.

Potain alludes also to the frequency with which amongst the infections paludism plays the part of an exciting cause. The same thing has been noticed with regard to that malarial disease, influenza. Cases have, I believe, been recorded where influenza ushered itself in by provoking a severe attack of asthma; I have seen such a case myself, and shall record it later in this article. But it is seldom, perhaps, that this is the case; far oftener asthma comes on as a result of the post-influenzal exhaustion of the nervous centres. I have notes of six cases of the kind.

Of nervous shock, or strong emotion, I will only add that, as such impressions are well known to bring on attacks, so they may also remove attacks instantaneously and completely.

In all these cases examine the patient in an interval of freedom, and there may be no evidence whatever of any disease. But of a large class of asthmatics this cannot be said. In many an asthmatic, for instance—80 per cent of all cases, according to Dr. Theodore Williams—there is evidence of permanent catarrh of the bronchial tubes (bronchitis). A little fresh accession of cold, and on comes asthma. Hyde Salter says much the same, namely, that 80 per cent of the cases of asthma in the young date from whooping-cough, bronchitis, or measles. There are other asthmatics who are gouty, and the gouty condition of blood seems to provoke a catarrh, in this respect occupying an analogous position to ague. The alterations of the ribs in old age lead to pulmonary obstruction and emphysema, and so favour an asthmatic paroxysm. The pulmonary congestions of chronic heart disease and renal disease bring about the same end. Hyde Salter describes a peptic asthma due to indiscretions in diet; but this seems to me to belong more properly to the idiopathic group, the stomach being a common point in the morbid circle from which the storm is set agoing. In many even of these secondary cases, however, it is still supposed, as I said at the outset, that some constitutional element or weakness allows the local disease to start the train of peculiar symptoms.

There is, however, one group of cases to which I am not sure that this applies. Every now and then an asthmatic appears to have suffered, for the first time, in middle life, in whom there are no obvious tendencies to neurotic ailments, and no evidence of existing disease that might act as proximate cause. It is possible that a percentage of these may belong to the group already mentioned, where gout in the system or excessive vascular tension has been the cause; but I am not satisfied that these things explain all the cases of later appearance. To judge from my own experience, they are prone to be very severe, and to be but little amenable to treatment; and I have come to the conclusion that in certain cases there may be some rapid onset of emphysema, some process of degeneration in the tissue of the lung, such as was described some years ago by the late Dr. Greenhow.

Simple spasmodic asthma is very seldom seen in the wards of a general hospital. It is of course found often enough in the degenerate, in association with emphysema, chronic bronchitis, gout, and renal disease. But in the primary pure form it occurs seldom indeed. There are many reasons for this. Chief of them, perhaps, is that this disease comes and goes; and for maladies of that kind the working-man cannot afford to lie up. Indeed this applies to all classes of society. As Berkart truly says, "Asthmatics are not disposed to consider themselves as patients. Their suffering is forgotten as soon as it is over." But I cannot help thinking that the affection is one that belongs more peculiarly to the upper ranks of society. It may be, perhaps, that the angular condition of the nervous centres, to which the disease may be attributed, becomes rubbed down, so to speak, by the harder life of the labouring classes; just as such persons are less sensitive to noise, less sensitive altogether to what one may call the smaller ills of life.

Results of asthma.—When a man has been the subject of asthma for a long time, it is likely that he will present characteristic appearances in his general physique and gait. He is usually very thin; his back is rounded, his shoulders are high, and he walks lethargically, with a well-marked forward stoop. He sits, may it be said, turtle-like, with his neck dropped into his chest. In long-standing cases the face is a little dusky, the eye watery and perhaps congested; and there is often a cough of peculiar timbre, moist and hollow, not easy to describe, but evidently the product of feeble expiratory power. The asthmatic speaks, too, as he coughs; and for the same reason, that the tidal wave of the chronic asthmatic is exceedingly shallow: for the spine is rounded, the ribs stiffened and fixed, the chest elongated and depressed.

The morbid anatomy of asthma, saving perhaps one particular detail, is comparatively small in amount and simple in kind. It is obvious that all diseases, as they fall under the denomination of "functional," must proportionately be wanting more or less in those coarser changes in structure that we look for in the study of morbid anatomy; and so it is here. The leading departures are most of them certainly conditioned by, and secondary to asthma of long standing: they are the results of the impaired respiration, not the cause of the asthmatic paroxysm. These are more or less chronic inflammation of the bronchial tubes, shown by injection and thickening of the mucous membrane, thickening of the muscular coat of the bronchial tubes, dilatation of the tubes, emphysema of the vesicular structure, more or less thickening and atheroma of the branches of the pulmonary artery in the lung, and hypertrophy and dilatation of the right side of the heart. The changes in the skeleton that go with these have already been mentioned; these are the curved dorsal spine, the barrel-shaped chest, the stiffened ribs, the generally wasted frame.

But we have still to consider in more detail the state of the bronchial tubes, and the products that are shed from their mucous surface.

The most regular condition to be found in the asthmatic is more or less mucus or muco-pus in the smaller tubes. This may be considered perhaps to be a feature of the asthma itself, inasmuch as it is admitted that in 80 per cent of the cases there is some organic change in the lung.

Practically, all the controversies that have been waged over this subject have centred in this: which is cause, and which is effect? Medicine, so eager to find a cause for everything, is unwilling to accept anything as such that does not possess a definite basis of structural change visible to the naked eye or to the microscope; and is willing to attribute the phenomena to any change that is demonstrable, rather than incur the suspicion of going beyond the facts, of hasty generalisation, or of appropriating prematurely the possessions of the future.

From a very early time Lefèvre, himself an asthmatic as recorded by Berkart, had described the expectoration of a peculiar kind of sputum; but we may take up the matter at the later date when Curschmann re-observed and redescribed peculiar elongated plugs or spiral bodies in the expectoration of the asthmatic, to which he was inclined to attribute considerable importance. To the formation of these bodies—"Curschmann's spirals," as they have been called—spasmodic asthma has been attributed. Their nature is yet in doubt, some considering them to be inspissated epithelium, some fibrinous concretions from the smaller bronchia; I should myself suppose, although this view is combated by Curschmann, that in their nature they are allied to the plugs that form in the so-called plastic bronchitis; and that like these—although their formation is an acute process—they leave behind them some habitual morbid condition of the bronchial tubes, and possibly also some proclivity to the formation of such bodies in the individual attacked. There is no doubt, moreover, that although more common in asthma, these bodies are found in pneumonia and œdema of the lungs also; and in certain secretions from the conjunctiva (Gerlach). Altogether it seems impossible to consider their presence as satisfactorily explaining the onset of a condition so peculiar as that of spasmodic asthma.

As little can be said of the Charcot-Leyden crystals present in the sputum and interior of the spiral plugs, and likewise of the eosinophile cells (Adolph Schmidt) in the blood and sputum of the asthmatic. I should agree with Müller and with Schmidt that the discovery of either of these bodies in the sputum does not definitely indicate the nature of the disease from which the patient is suffering: if this be so, the contention that they have any causal importance has little to support it.

Such, then, is the history of an attack of asthma; such, and it is but little, is the morbid anatomy that is associated with it. But these, and certain physiological experiments, are almost the only data upon which to frame a pathology of the disease.

Pathogeny.—A disease that is, or may be, so sudden as to be well-nigh instantaneous in its onset—one produced under the influence of strong emotion, one which, under such and other circumstances, may subside as quickly as it came—can hardly be other than some functional aberration of normal structures. The changes that seem most competent to explain the phenomena of asthma are: (i.) A muscular spasm of the smaller bronchia; (ii.) Some rapid tumefaction of the mucous membrane of the bronchia; (iii.) A rapid production of collapse of parts of the lungs. All these, as has already been said, are hypothetical causes only, although each one of them can be defended by more or less cogent arguments. The one most generally accepted of recent years, it need hardly be said, is that the production of asthma is due to spasm of the muscular coat of the smaller bronchia. The late Dr. Hyde Salter, who was the chief and most able expositor of this view, makes use of the following arguments in its favour:—

"In the first place, the sudden induction and remission of the asthmatic paroxysm is consistent with this supposition; in the second place, there is abundant proof that the air in the lungs is locked up, and can neither be got in nor out; there is evidently plenty of air in the chest, percussion is even over-resonant, yet the patient is as unable to drive air out as to draw it in; he can neither inspire nor expire; he cannot discharge breath enough to whistle, to blow out a candle, or to blow his nose. The muscles of respiration tug and labour to fill and empty the chest, but the chest walls remain almost immovable; the inspiratory muscles cannot raise them, the expiratory cannot depress them. On listening to the chest we find corroborative evidence of the stagnation of the air. The respiratory murmur is in a great degree lost. This absence of respiratory sound, accompanied by violent respiratory effort, is one of the most striking and suggestive of the facts of asthma. How can we explain it except by supposing that there is some bar to the ingress and egress of air; and what can this bar be, unless it be spasm of the bronchial tubes? It cannot be inflammatory thickening of the mucous membrane lining them; for the sudden, almost instantaneous establishment and remission of the dyspnœa is incompatible with this. It cannot be mucus plugging the tubes, for the attack will often come and go without any expectoration whatever. But we have still more positive and precise evidence of the circumscribed narrowing of the air-tubes in the musical sounds that are present in asthmatic breathing. This symptom has all the certainty and precision that characterise physical phenomena; and it shows that the air-tubes are the seat of constrictions which throw the air passing through them into vibrations, and convert them into musical instruments: since these musical sounds are multitudinous, the points of constriction must be many; since they are constantly varying in locality and character, the constrictions of the tubes must be undergoing similar change. Lastly, the effects of remedies and their nature tell the same tale, and point to muscular spasm as the immediate essential condition. The most powerful remedies for asthma are what are called cerebro-spinal depressants, such as "emetics, tobacco, etc., remedies the direct effect of which is to relax muscular spasm" (13).

This view, originally affirmed by Reisseissen, who based it upon his discovery of the presence of circular muscular fibres in the bronchial tubes, has since then been proved on experimental observation by numerous observers from C. J. B. Williams onwards, including Paul Bert, Riegel, Biermer, Lazarus, and others.

This spasm is held sufficient to explain a state of things round which a good deal of controversy has centred, namely, the over-distension of the chest that occurs in the asthmatic, associated with a dyspnœa that is mainly an expiratory one. The obstruction in the tubes being incomplete, it is said that the air, under the labour of forced inspiration, enters the lungs, but that expiratory paralysis or obstruction prevents its getting out again. It is objected, however, by Wintrich and others, that as the expiratory force is greater than the inspiratory this solution is not satisfactory. The lungs under such circumstances should tend to collapse. Wintrich, accordingly, believes the attack to depend upon spasm of the diaphragm. But this explanation does not seem free from difficulty; for, as Wilson Fox says, the phenomena of "this condition are widely different from those observed in spasmodic asthma."

No doubt if one is to accept as absolute that doctrine which teaches that atelectasis is a necessary consequence of the collection of mucus in tubes that narrow progressively from the trachea to the periphery, because plugging of such tubes creates an inspiratory difficulty more than an expiratory, the air being able to get out of such tubes under the pressure of the expiratory force, but hardly to get in under the ordinary inspiratory act,—there is a difficulty. The distended chest of asthma contravenes the usual rule.

But the asthmatic state is a complex one, and it is to be explained by no simple and universal law. In the first place, granting the existence in bronchitic states of conical tubes and adapted conical plugs,—which, after all, is an imaginary description,—a spasmodic contraction of the bronchial muscles is not the same thing as a plug of mucus in the tube. In the one case the obstruction might, and probably would, to some extent be on the side of expiration; it could have no such effect in the other, unless indeed the spasm were regular and rhythmical from the periphery upwards towards the main tubes. A spasm of the tubes must tend to prevent air getting both in and out; and the more in or out according as the inspiratory or expiratory force is the greater. But the expiratory force is said to be the greater, and so it may be for ordinary respiration, but no one who has seen the forced action of the ordinary and extraordinary muscles of respiration in the exceeding labour of inspiratory effort during an attack of asthma, can have any doubt that the natural order is completely destroyed, or have any difficulty in believing that the air is really sucked past the obstruction, so that the lung becomes over-distended. Nor is it unimportant in this regard to insist again that the obstruction flits about from one part of the lung to another; from one side to the other; a temporary relaxation of spasm which means a liability to compensatory over-distension of the unlocked part.

Some authors, unable to get over the difficulties which this assumption of spasm of the bronchia creates, have suggested a spasm of the respiratory muscles; others, again, a paralysis of the muscles of the bronchial tubes. As regards the latter, admitting that under ordinary circumstances the muscular coat of the bronchial tubes may be reckoned as one of the forces of expiration, it is hard to think that its share can be so great that its failure should constitute a departure from normal so grave as asthma. More might possibly be said in favour of a spasm of the muscles of respiration, for, if we run over the field of clinical medicine, we are not unfamiliar with several curious vagaries of breathing which are attributable to such a cause: Cheyne-Stokes respiration is one of these; the air-hunger of heart disease another; the asthma of uræmia another. All these, in common with spasmodic asthma, are immediately conditioned by some convulsive or misapplied action of the respiratory centre, and perhaps give some colour to the suggestion that one of the factors in the production of spasmodic asthma may lie in aberrant action of the muscles of the thoracic walls.

It must be admitted, however, in the present state of our knowledge, or of our ignorance, that, although other explanations may seem to some to be as good, the theory of muscular spasm is at any rate fairly complete. It is a reasonable and satisfactory explanation of the facts, and it does not appear that there is much that is convincing to be said against it. Nevertheless, the hypothesis placed second in order is, I think, almost equally good, that which assumes some rapid turgidity or erectility of the bronchial mucous membrane. The capacity of such active congestion, even in parts that are not naturally erectile, is well seen in certain morbid phenomena—in some cases of Graves' disease, for example, where from some sudden shock the eyes have as suddenly become prominent; that this is a purely vascular turgescence is shown by its complete subsidence after death, and by the fact that the orbital or ocular structures show no morbid change: now if we look to the respiratory tract itself we all know only too well how near a common cold may come to an attack of asthma. There is the initial irritation of the nostrils, then the sneezing, then sore or dry throat, then some little tracheal worry, and finally a definite, albeit slight, bronchial stuffiness and wheezing. Now in these cases the initial change is certainly turgescence of the upper air-passages; and so also is it in the cases of paroxysmal sneezing, and in certain cases of local disease of the nasal mucous membrane, of deflected septum, or of polypus.

It seems, therefore, a rational belief that what can be proved to exist in the upper air-passages, namely, a definite erection or turgidity of the nasal mucous membrane, may also take place lower down in the bronchial mucous surface; and, so doing, accomplish much of what we call spasmodic asthma. It may be thought, however, that swelling such as this is hardly adequate to explain the sudden origin and subsidence of the paroxysm; yet it is quite competent to do so, for paroxysmal sneezing comes on quite suddenly, and subsides again as readily if, from any cause whatever, the attention be averted from the subjective discomfort. This hypothesis also is not without a considerable body of influential support, dating too from early times. Wilson Fox states that Bree, as early as 1807, held some such opinion, considering that asthma was a convulsive attempt to expel peccant material from the bronchial tubes. Traube considers asthma as a very acute catarrh. Blackley contends that the asthma of hay fever is the turgescence in the nose extending to the general bronchial mucous membrane. And Sir Andrew Clark considered the phenomena of an attack to be explained by a vaso-motor neurosis, by which changes analogous to those of urticaria upon the skin are produced.

To this it may be added, that Störck actually observed with the laryngeal mirror that in certain instances of asthma the whole length of the trachea and part of the right bronchus were deeply congested.

Berkart, however, will have nothing to do with a neurosis of any kind. Although he admits the existence of a peculiar predisposition, he will not allow that the history of the asthmatic attack, as regards its sudden onset and sudden subsidence, is anything but vague and untrustworthy report. He sums up his opinion thus: "The conclusion, therefore, seems irresistible, that what is commonly described as bronchial asthma is an acute and progressive, nay almost erysipelatous, form of inflammation, which extends from the pharynx upwards and downwards, and is accompanied by a croupous exudation." But it is impossible thus to discard so large a body of evidence, vague though some of it admittedly be, as case after case of asthma supplies. And, if not, then the surmise of an initial inflammatory process is much less securely seated. Indeed, "an acute and progressive, almost erysipelatous form of inflammation" may well be thought to land its advocate in even greater difficulties, seeing that the disease is seldom associated with fever, seldom with any pneumonia, and, as an attack, is never a cause of death.

I believe, on the contrary, that the explosive character of asthma is absolutely certain. Let two cases suffice:—A gentleman was seized with influenza, and it was ushered in by an attack of asthma. This condition was supposed to have gone on to broncho-pneumonia, and this proved intractable. At great inconvenience to himself, therefore, he obeyed the order to take a long holiday; and it was my good fortune to see him as he passed through London, within two or three hours of his leaving home. I was to find a particular focus of disease at an indicated spot. The man told me, as so many asthmatics do tell us, that he felt his breath relieved after he had been twenty minutes in the train; and when I saw him an hour or two later, no one could say that he was other than absolutely healthy. The second case I owe to the editor of this work. Dr. Allbutt was examining by auscultation the backs of the lungs of a gentleman of neurotic habit, who was overworked and suffering from pains which were suspected to be of the nature of angina pectoris. The patient was sitting up in bed, and his face was under the observation of his own medical attendant. After hearing a few inspirations of a normal character, to Dr. Allbutt's surprise the inspiratory murmur began to diminish on the left side, and in a few moments ceased. Perplexed by this strange event, percussion was quickly applied to the left side of the chest, but with negative results. During this time the family attendant, Mr. Bowman of Ripon, saw the patient striving for breath; and attention being drawn to his state, it became manifest that he was in his first attack of asthma: respiration quickly became almost inaudible over both lungs, and then, after a definite interval, sibilus supervened. The attack followed the usual course, and the asthma thereafter frequently recurred.

As regards the sudden occurrence of atelectasis pulmonum, alleged as a cause of the disease, there is perhaps less to be said. Nevertheless, it may be well to point out that, in infancy at any rate, there are cases, and these by no means infrequent, where atelectasis occurs very suddenly; and there are cases where the auscultatory evidence makes it probable that this condition flits about the lung in a manner almost comparable to that of the migratory passage of the asthmatic paroxysm. And for my own part, I believe it to be probable that acute collapse of the lung occupies a much more important place in the production of pulmonary affections than is supposed.

Diagnosis.—It is not necessary to linger long upon this section of the subject. It is true that many affections are called asthma that are not so regarded in this article. The short breath and the dyspnœa of chronic bronchitis, and the dyspnœa and orthopnœa of heart disease, are often thus designated; in both of these the dyspnœa is rather a subdued distress than the acute agony of spasmodic asthma; so with the air-hunger of some cases of renal disease, and of dilatation of the heart. The inspiration is free in such cases, but panting; it is not a dyspnœa. And yet it must be added that sometimes, in the early history of a granular kidney, the complaint of the patient may be chiefly of asthma of a mild kind; and without a general investigation of the case, without the hard pulse, the thick first sound, and perhaps the retinal changes, the real nature of the case might be overlooked. Of other conditions more likely, perhaps, to give rise to mistake I incline to place hysteria. I have certainly found myself occasionally in a difficulty between the one and the other, more particularly when it has been necessary to depend upon the history of the attack as submitted for an opinion, some time, it may be, after all the symptoms have passed away.

I am reminded also by the editor, first of a restless disturbed sleep, experienced by some persons, that is really a mild asthma, although not recognised as such by them; secondly, of that curious faucial or laryngeal suffocative spasm, often in gouty people, that awakens the subject of it in the middle of the night in terror lest he should choke.

Mediastinal tumours and aneurysm of the aorta, by leading to paroxysmal dyspnœa of a sort, are sometimes liable to be overlooked in a hasty diagnosis of the more familiar disease. And there are various obstructive maladies in the upper air-passages that may, in like manner, cause difficulty at times. The safeguard against mistake lies in the unfortunate fact that asthma is very common, and therefore in its usual features is very generally known; and in respect of other maladies, even should they be entirely paroxysmal which is uncommon, each one has usually some peculiar feature of its own that is sufficient to arrest the attention. Any one of these things may, of course, exist in association with the special nervous proclivities of asthma, and it might then become a matter of the greatest difficulty to distinguish between the morbid occasions of the spasm. Nevertheless, it may be doubted whether in practice this difficulty often arises.

Treatment.—We will first consider the principles and afterwards the details, lest in the multiplicity of the latter, and in the urgency and intractability of the disease, we lose our hold on the principles to which details ought to be subordinate. As I have already said, to me it seems impossible to doubt that asthma is one of those nervous actions of which we see so many examples in our economy, and which have been well called by Dr. Edward Liveing paroxysmal neuroses.

Epilepsy is one of these; some forms of insanity are others; migraine is another; asthma is another, and so on. Now all these more or less obey this law, that the more they come the more they may. Nervous actions, which in their essence and initiation are not abnormal, by excess of energy, or of frequency, or of both, become abnormal; and ultimately a bad habit becomes fixed. Surely, both in epilepsy and asthma there is much of habit in the intractability of the disease; and if control is to be gained over either, it must be by catching it in the earlier days of its appearance, and by arresting it before it becomes confirmed. We think that we can sometimes gain some control over the convulsions of infancy; we can perhaps keep them at bay sometimes, and so stop the child from becoming epileptic. But what case is more hopeless than that of the confirmed epileptic, even though he be persistently stupefied with bromides?

The case of asthma is a parallel one. It has been contended that it is a disease rather of childhood than of adult age; and that to pay attention to this fact, and to the suggestions that flow therefrom, offers the best possible chance of stopping the attack, and of preventing the fixation of the habit and the establishment of chronic asthma. The chronic asthmatic is almost as hard to cope with as the chronic epileptic.

There are two methods of dealing with the asthmatic. On the one hand, we may attempt to make the environment of the patient conform to the conditions required by the individual; or, on the other hand, to harden the individual, to widen his range of accommodation, and so to make him less susceptible. And in the matter of drugs somewhat similar alternatives present themselves; we may either give sedatives to the over-sensitive nerve structures concerned, or give drugs, if such there be, to raise the level of nervous action to that higher platform that shall enable the perceptive centres to take less heed of their unnatural worries.

But the asthmatic paroxysm is so distressing that, almost always, the treatment of it usurps the first place; and too often this urgency of the situation upsets the perspective. If we are called to a patient in the stress of a paroxysm of asthma, clearly, on all accounts, it must be arrested as quickly as may be; there is no time to be very careful and consistent about ways and means. And the quickest way to relieve a paroxysm of asthma is to make the patient inhale some fume or other, as of nitre, nitrite of amyl, or chloroform; or to give him an injection of morphia or a dose of chloral; indeed, as we all know very well, doctors see paroxysms of this kind less often, because various patent powders for creating fumes hold the field so largely that most people do without us, and stick to their patent remedy.

Thus the treatment of asthma too often becomes a repeated sacrifice to the paroxysm; and the patient drags along, thankful for the small mercy of temporary freedom from his troubles, and easy in mind if he can carry in his pocket protection from those that are to come. But this plan of campaign is ultimately a most disastrous one. It unquestionably produces temporary ease; but what happens afterwards is this: the vapour, on reaching the mucous membrane, stupefies or exhausts the nervous centres, and stops the spasm for a time. But at the same time some of these remedies, by stimulating the mucous membrane and provoking the flow of mucus, make the local erethism rather worse than it was before. The more sedative kinds of inhalations do but appease by offering bribes to vicious nervous influences. By and by the nervous centres wake up again to find matters no better, rather the contrary; and then on comes the spasm again, and the whole process is repeated; and, with each repetition of the cycle, the nervous centres, as their nature is, become more exhausted or more irritable, their sleep is shorter, their spasm is more and more quickly repeated, and the poor patient ultimately lands himself, with perhaps some lessening of the severity of each paroxysm, in a more prolonged or persistent stuffiness hardly less distressing to bear: all day long he appeals to his powder, and becomes in fact the slave of an appetite that he has whetted and that he cannot now control. Thus ends the chronic asthmatic who betakes himself to vapours. But this is not all, for by common consent a repeated application to some of these drugs, whether by making matters worse in the lungs, or by worrying the cardiac ganglia or what not, tends to dilatation of the heart, and is equivalent to a good many nails in the coffin of the asthmatic. Moreover, this dread of the paroxysm itself is carried into the preventive treatment of the disease, and the patient is submitted to what may be called the glass-case treatment; that is to say, the temperament of the patient is ignored, or not considered as of importance, and the disease is supposed to be brought on by chill. If he be wealthy and adventurous, he fights his environment by running away; and thus he may, perhaps, get along pretty well. If the patient be a child, it is probably kept indoors, except in the finest of summer weather; yet, nevertheless, the history too often is that "it has caught another chill," but no one can say how. At first, perhaps, the child had the whole house to roam about in, but, as the "colds" recur, it is confined to one room with a south aspect; and yet things do not mend. So the doors of the room are carefully screened, the windows perhaps pasted up, and still the success being not all that can be desired, extra clothing may be piled on. And ultimately the doctor finds somewhere hidden under this heap of precautions a pale, moist, flabby, steamy thing, with big eyes, thin cheeks, protruding ribs, and a more or less general bronchitis; a case of "successful" management, because no attack has occurred for some weeks! But is this to be called success! This is to nurse the powers into imbecility; and the inevitable result is, that the first time the patient puts his head outside the door a fresh cold is "caught," and a fresh term of imprisonment is ordered. I venture to say that if asthma is to be prevented at all, it will never be kept at bay by hot-house treatment such as this. Yet, unfortunately, it is easy to utter destructive criticisms of this sort, but difficult to point to a better way. I think there can be no doubt that the first requirement for the asthmatic is to put him into a climate in which he can be much out in the air. But there is the difficulty: we know so little about climate; and asthma is so individual a disease. No one can foresee in a particular case whether this place or that will be suitable; and, when the issue is doubtful, experiments in moving invalids about are never likely to be made with any great thoroughness. But for most asthmatic persons there is generally for each his own place or places where he is better or well. Thither he should be sent, at any rate, for a time. This place may be at the sea; or it may be inland; sometimes it is a dry place, sometimes a humid; often even it is a large town: "In the great majority of cases an urban air is the air that cures; and of a city air, that seems to be the best which is the most urban—the densest and smokiest" (Salter); but wherever it be, the patient should be out and about with very little restriction; and an attempt should be made by this means to render the morbid circuit less prone to discharge. Of games and sports, all should be encouraged that are outdoor and healthful and invigorating. Some further remarks on climate in the treatment of asthma will be found in the first volume of this work (p. 293).

In diet it is necessary to be careful, but not too much so. It is very easy to give a number of restrictions about food, and thus to make matters worse; yet asthma certainly often does seem to start from a meal that has not been digested—one which may have been too large, of an improper character, or taken at some irregular hour: the points to aim at are good, plain, light food in moderate quantity and slowly ingested. The asthmatic, particularly children, are often deprived of potatoes, of starchy puddings, jam and sugar, and goodness knows what else, and on the other hand are put on various meat juices and other good things in the wrong place, so as to remove all rocks of offence from the path of their pneumogastrics. But "if these things be done in the green tree, what shall be done in the dry?" What chance has such a child of reaching old age? Any food that is plain and wholesome and not known to disagree may be allowed. It is a good thing to have the chief meals early in the day, when digestion is vigorous; therefore breakfast and lunch—an early dinner—should be the main meals; anything taken later must be small in quantity and of the most digestible kind. All meals for the asthmatic should be small ones; his stomach should never be distended [*vide* art. on Dietetics, vol. i. p. 398]. The bowels should be kept carefully regulated and sufficiently open by taking some saline aperient, or other simple laxative. Every effort should be made to keep the patient in as healthy and physically fit a condition as possible. A tepid or cold bath should be taken in the early morning, and the living room well ventilated.

These must be the general principles upon which to deal with the asthmatic; and the more unhesitatingly the younger the patient, and the earlier in the course of the disease that he comes under treatment.

In considering the treatment by drugs, two divisions of the subject naturally suggest themselves; namely, those medicines that are useful in preventing asthma, and those that are so when the actual attack is threatening or in progress. Again, a distinction must be made between the cases which seem to be pure nervous asthma, those which have any degree of persistent bronchial catarrh, and those already mentioned, which come on in later life, and may not irrationally be attributed, on the one hand, to blood conditions that as a group may for convenience be called gouty, and, on the other, to degenerative changes in the tissues.

As a preventive remedy in the pure form of asthma, no drug is in my opinion equal or nearly equal to arsenic. It should be taken for three or four weeks, then omitted, and then resumed after an interval of equal length; and so on for three or four courses: and the drug should from time to time be resorted to in periods when from any cause the nervous centres begin to show signs of lowered tone. I have not made much use of phosphorus, but it has been spoken well of, and it might also upon occasion be of value; and so likewise with other good nerve tonics, such as bromides or hydrobromic acid.

In cases where a persistent bronchial catarrh is at the bottom of the trouble, there is obviously less to be expected from medicine, and a suitable climate promises best; as a rule such cases do best in dry and bracing air. A friend of mine thus circumstanced found himself almost renewing his youth as he climbed the Malvern Hills. Others again find more relief in such places as Hastings, Ventnor, Bournemouth; some even in Torquay. Good results are claimed in the bronchial cases for the sprays and waters of the sulphurous springs of Mont Dore, of the Pyrenees, of Harrogate, and of the arsenical waters of La Bourboule. The two chief drugs from which much benefit is often derived are strychnine in three to five-drop doses given steadily for some days, and the iodides which often prove of great value. Perhaps the one acts as a stimulant to the respiratory centre, the other as an expectorant. For the asthma that occurs in later life an eliminant treatment is on the whole the best. It is in such cases that blue pill and colocynth in moderate doses once or twice a week are useful, or saline laxatives with careful attention to and restriction of diet. In these cases, again, iodide of potassium, perhaps by a depressing effect upon the arterial pressure, will often help very considerably.

My friend Dr. Kingscote maintains that the asthmatic state is much benefited by brine baths and systematic exercises, such as have been elaborated at Nauheim, for the treatment of certain forms of disease of the heart; one can well understand that means of this kind, by stimulating the circulation and facilitating the flow of blood through the lungs, may prove of much service.

To relieve an attack, or the semi-asthma that forebodes or lingers after an attack, other means must be used. In the threatening of an attack, or in the dyspnœa that lingers when the more acute symptoms have subsided, many drugs have been tried, and at one time or another succeeded. Of these I should put first a combination of iodide of potassium with the etherial tincture of lobelia; five, ten, or even fifteen grains of the one, and ten or fifteen minims of the other, seem to bring relief when other things may have failed. Some prefer stramonium to the lobelia. The late Dr. Hyde Salter thought very highly of the Datura stramonium and the D. tatula; their best effects are observed when smoked like tobacco; but they may also be given in a pill, extract, or tincture. Sometimes a combination of iodide of potassium and chloral hydrate has been effectual. It is under such conditions as these that the Euphorbia pilulifera and Grindelia robusta are most useful; the former may be given in a decoction, a wineglassful twice a day; or in tincture, ten to thirty minims, twice or three times a day, or as often as may be requisite. The grindelia is in the form of a liquid extract, and is given in similar doses to the tincture. This drug is also recommended at the onset of an attack, in half-hourly doses, until relief has been attained. I have known it to produce decided relief, but I have not, upon the whole, been very successful either with this drug or with euphorbia. In the thick of an attack the remedies most in use are inhalations of various vapours; and of these, perhaps the commonest, and one of the most harmless, is blotting-paper soaked in nitrate of potash, which will often relieve and sometimes very conspicuously.

There are many other powders made for the production of fumes; some are stimulating, and seem to act by provoking cough and the free secretion of mucus; others, and these I believe the less harmful, are of a sedative nature. Some of them are made into cigarettes for smoking, and most of them contain stramonium in some form.

Of inhalations available for more strictly medical uses, three may be mentioned: nitrite of amyl, iodic ether spoken well of by Dr. Thorowgood, and of course chloroform. In severe cases the last named may be of the greatest possible value, although its effect is apt to be but transitory, and the attack may resume its severity as the stupor of the drug wears off. Of all the other drugs that have been recommended for the relief of the paroxysm, morphine probably stands first; a hypodermic injection of a sixth of a grain will often procure almost immediate diminution of the violence of the dyspnœa, which gradually ends in complete cessation of the spasm. Pilocarpine is also a valuable drug; a tenth to a quarter of a grain may be given hypodermically; a free secretion from the mouth and fauces is the result, and the spasm is thus relieved. Sometimes the patient is sick, a thing by no means undesirable; for an emetic is one of the means advocated for arresting an attack, and no doubt sometimes with marked success. A combination of bromide of potassium and chloral is also a good sedative to give at the onset of a paroxysm. Belladonna, hyoscyamus, and conium, though not of so much value, are all of use in their way; tobacco is also said, by virtue of its powerful depressing action, to be a useful palliative drug. I have heard it said of pilocarpine that the remedy is worse than the disease; and, considering the distressing nature of the malady, this is a serious attack upon the benefit derived from it. If this be true as regards pilocarpine, it must be still more apt for tobacco, which produces a dreadful malaise, and is a difficult drug to control in those who are unaccustomed to its use, in whom only it appears to have the effect wished for.

Of stimulants, too, coffee and alcohol may be mentioned. Strong coffee is indeed a popular remedy that has often given relief, as also has citrate of caffeine. As regards alcohol, I have no personal knowledge of any special virtue, but Hyde Salter says of it, that while in many cases it does not do much good, in some it has a most powerful effect, particularly when all other remedies have failed. It should be given hot and strong. The compressed air treatment of asthma is described in the first volume of this work (pp. 315, 316), to which the reader is referred.

Hay fever, or hay asthma, is in the opinion of many a pure form of asthma, and with this opinion I myself coincide; it is accordingly more or less amenable, as are other forms of asthma, to treatment by drugs of the same character. I refer more particularly to arsenic; and I should maintain this even for nasal cases: it relieves the itching and smarting of the eyes, the aching of the frontal sinuses, the itching of the nasal mucous membrane and of the nose itself, the sneezing, the watery discharge, the occlusion of the nostrils, the dryness and irritation of the lips and throat. But Karl Binz and others have maintained that local remedies, used upon germicide principles, give great relief in many of these cases; and those who have worked in the special department of diseases of the nose and throat declare that, by paying special attention to the morbid erectility of the mucous membrane over the spongy bones of the nose, this disease may be much reduced. Binz advocated the irrigation of the nostrils with a solution of quinine; Sir Andrew Clark suggested some carbolic preparation; and of late many have tried the application of solutions of cocaine, more upon alleviative than upon curative principles, perhaps; unless alleviation be an earnest of cure.

No one can doubt that these various measures are all useful in their proper place, nor can any one doubt that they have their dangers. For instance, I saw but the other day a lady who for the discomfort arising from the frequently recurring turgidity of the nostrils, which is characteristic of hay fever, had betaken herself by medical advice or without it to the use of cocaine locally. Accordingly, more or less, both by day and night, she would pack her nostrils with a solution of cocaine, of which one grain at each time was put into each nostril; and thereafter, by means of hawking and spitting, and other contortions of her pharyngeal muscles, the solution was spread all over the affected area, and temporary ease was obtained. At least six grains a day were thus disposed of, and sometimes more. The position to take with regard to local treatment is this, as it seems to me: the local symptoms are not the disease, and therefore, however necessary it may be at times to relieve conditions that cause great distress by means of this kind, they may do harm by inducing other morbid changes in the part, and conditions that were but temporary may thus be rendered permanent. For instance, a paroxysmal sneezing will stop immediately under the influence of some diverting train of thought, just as asthma will stop under any sudden and powerful mental stimulus. All must agree that if there be any actual disease of the mucous membrane, whether due to the existence of polypus, of a deflected septum, or what not, it must be advisable to get the mucous surface into as healthy a state as possible so as to remove one obvious source of possible irritation. But for cases of asthma in which there is no definite nasal worry, the question must at any rate be considered an open one. If the neurotic origin of the disease be accepted, as I think it must, no one can rest content with the treatment of a peripheral symptom. Still, if it can be shown that a large measure of relief is thus obtained, such an experience must of course be utilised upon the principle that half a loaf is better than no bread. The difficulty of arriving at any sound conclusion as to the value of such treatment lies in this, that the specialist and the physician see the cases at different times: the one in the first flush of that post-operative quiescence that we all so well recognise as a characteristic of nervous ailments; the other, when that quiescence has passed off, and the old habit has resumed its sway. I have known some patients to be apparently benefited, but others who have not received any adequate reward.

Prognosis.—I hold most strongly that asthma may be treated with a large measure of success if it be taken in hand at the proper time, that is in childhood; and if it be possible to put the patient under suitable conditions,—those conditions being, in brief, such as will allow of the patient being turned into a good healthy animal. It is in childhood, if at any time, that the opportunity offers of educating the patient out of a faulty habit into a better regulated state of his nervous centres. In the case of the adult one cannot be so hopeful. One could not be so hopeful of successfully combating convulsive attacks occurring late in life as in those occurring in infancy; and I fancy that the asthma that begins in adult age is indicative of some deep ingrained nervous fault, which is not readily to be controlled. Moreover, adults fall in less readily with counsels of perfection, such as the radical cure of faulty habits; they are in distress, they insist upon a dose to set them right, and if one man won't give it them, or does not hit upon the right thing, they quickly resort to some one else, who manages things, as they think, better. Still, even the adult asthmatic is sometimes a sensible person, and many agree to desist from inhalations; to take a drug, such as arsenic, patiently; or iodide, when an attack threatens; or such other drug as may seem best suited to the particular case: to act thus is in most cases to procure considerable relief.

With regard, however, to that other group, when spasmodic asthma occurs in middle age, and after; when, as causes or provocatives, certain changes in the tissues and organs, gouty and other, come into prominence; and when age with its paling vigour of function and its conscious or unconscious indiscretions of living and other habits leads to the overcharging of the blood with waste products, and to excess of arterial blood-pressure, and thus to a true spasmodic asthma analogous to the gouty vertigo and gouty convulsion occasionally seen in adult life: then no doubt great relief, and even cure, may result from such drugs as blue pill, iodide of potassium, and others, given with the purpose of reducing the arterial pressure, or of eliminating waste products. More difficult to speak hopefully of are the cases associated with and perhaps produced by a pre-existing bronchial catarrh. As Hyde Salter remarks, we send such patients to the Riviera to relieve the bronchitis, and the asthma is aggravated; and thereby we see in a measure how essentially independent the two conditions are; and when, after travelling about, they come to the land of promise so far as their asthma is concerned, then perchance the climatic conditions are not suited to the bronchitic affections. But even in such persons there is no doubt that, by dealing with the bronchitis and by endeavouring to ameliorate it by means of a suitable climate, the bronchial tubes will become more healthy, and there may yet be scope for carrying out those principles of reinvigoration of the nervous tone upon which I have dwelt; and thus some of the stress of the asthma may be relieved. Nevertheless, when the best has been done, one cannot but regard the disease as serious, and in too many cases baffling; for even in cases where much good seems to have been done, the disease reappears again, perhaps after many years. In looking over notes of a number of cases, it comes out clearly that in several where the disease existed from, say, the age of three to ten years, it reappeared at forty or fifty. I have already alluded to the many points of similarity between asthma and epilepsy; and this is another feature of resemblance. We meet, too, with many people who have lost the tendency, and who are still free; but many of these, although they say they have lost the asthma, are still a little wheezy, and undoubtedly have some slight amount of bronchial catarrh. So that on the whole there is a degree of uncertainty about the fate of those who are asthmatic in early life. As regards the actual duration of life, perhaps all that can be said is that spasmodic asthma is compatible even with a long life. Of those who become asthmatic in later years, excepting the group of cases due to high arterial pressure already mentioned which may be a fairly large one, most are likely to suffer severely; and their disease is but too likely to become more or less permanent.

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SYPHILITIC DISEASE OF THE LUNGS

Our knowledge of the anatomical characters and clinical history of syphilitic disease of the lungs is still very incomplete, notwithstanding that much has been written on the subject. This is due in part to the rarity of the affection, but chiefly to the difficulty until lately experienced in distinguishing between the lesions of syphilis and of tuberculosis.

Up to the date of the discovery of the tubercle bacillus it was very often impossible to determine with certainty during life whether a given case of pulmonary disease were tuberculous or not; and after death appearances which some considered to be distinctive of tubercle were said by others not to possess this significance. Now, however, that we possess a test for tuberculous lesions, it may reasonably be hoped that the whole subject of syphilitic disease of the lungs will be placed upon a secure foundation.

That the disease is of rare occurrence is a fair inference from the fact that the museums of the London hospitals and of the Royal College of Surgeons, all of which I have recently visited, contain only twelve specimens which are believed to illustrate syphilitic lesions of the lungs; and of these, two may be excluded, as either not of that nature, or of a nature so doubtful that in the present state of our knowledge they are inadmissible as evidence. None of these specimens is from a case of congenital syphilis.

Morbid anatomy.—The following pulmonary lesions have been attributed to syphilis: (*a*) gumma; (*b*) white hepatisation (Virchow, Weber), or "epithelioma of the lung" (Lorain, Robin); (*c*) gray infiltration (Welch, Pancritius); (*d*) lobular pneumonia or broncho-pneumonia (Förster, Welch); (*e*) fibroid induration; (*f*) changes in the lymphatics (Hermann Weber); (*g*) a destructive disease, the so-called "Syphilitic Phthisis." It will be convenient to consider separately the lesions of the hereditary and the acquired disease.

Hereditary syphilis.—The pulmonary changes in hereditary syphilis may be either circumscribed or diffuse; to the former the term "gumma" is applied; the latter are classified under the head of "pneumonia." It is, however, far more common to find the two changes associated than to meet with either separately.

A. *Gumma.*—As this lesion is of comparatively rare occurrence in congenital syphilis, and when present does not differ either in appearance or in microscopical structure from that found in the acquired disease, a more distinct picture of the morbid anatomy of the two affections will be obtained by describing it under the latter heading.

B. *Pneumonia.*—Two different lesions are included under this heading—namely, "white pneumonia" and "interstitial pneumonia"; but of these it must again be stated that they occur more often in combination than apart.

(*a*) *White pneumonia* (Virchow, Weber), *Epithelioma of the lung* (Lorain, Robin).—This lesion, which in its true form is rare, is found only in the lungs of still-born children, or of such as have survived their birth a very short time. Other unmistakable signs of congenital syphilis are usually present, and in such cases gestation has seldom proceeded to the full term.

It is a diffuse change affecting a lobe either as a whole or in part; or one or both lungs may be completely consolidated.

In still-born children the affected part is bloodless and airless; even if force be used, it may be impossible to inflate it; but in infants several days old the lung always contains some air.

The lung is much increased in size, and its surface may be marked by the ribs. It is solid, dry, white, yellowish, or grayish white in colour; but sometimes presents a reddish marbled appearance. The section differs from that of an ordinary pneumonic lung in that the granular appearance characteristic of the latter is absent, the surface being smooth and somewhat shining.

On microscopical examination in true cases the interstitial tissue is not increased. The alveolar walls are thickened, and the small bronchi and the alveoli are filled with masses of cells of which some are round and others have more or less the character of epithelial cells: the cells are for the most part undergoing fatty degeneration and are beginning to break down. The alveoli are markedly enlarged. The colour of the affected area is due partly to the above changes, but in part also to diminished blood-supply the result of pressure upon the capillaries. The lung tissue surrounding the consolidated part may show some degree of emphysema. Ecchymoses may be present in the pleura, pericardium, and thymus gland; but these appearances are probably incidental to the mode of death.

The bronchial glands are as a rule enlarged and on section dense, from a new formation of fibrous tissue enclosing cells arranged in a concentric manner.

White pneumonia is a lesion of purely pathological interest, as, owing to the filling of the alveoli with cells, the subjects of it, if not still-born, are unable to maintain the respiratory function for any length of time, and soon succumb.

(*b*) *Interstitial pneumonia.*—This is the most common pulmonary manifestation of hereditary syphilis; but it occurs more frequently in association with some of the changes described under "white pneumonia" than as a purely interstitial lesion. In its true form it is distinguished by a small-celled infiltration of the interalveolar connective tissue, the alveolar epithelium remaining unaffected. This change may be present to a very varied extent. In some cases lungs thus affected appear normal to the naked eye, the lesion being only discoverable on microscopic examination.

In well-marked cases the lungs are large and hard and of a pale or dark grayish red tint. The change may be present throughout the organs, or a single lobe or portion of a lobe may be alone affected. To the naked eye the lung tissue presents a decidedly coarse appearance. On microscopical examination a marked increase is seen in the interalveolar and interlobular connective tissue, which forms broad meshes including small spaces wherein the alveoli are either crowded together or completely obliterated.

In some cases the interalveolar meshes appear to consist of a dense capillary network, the vessels being dilated and tortuous. Around the vessels and bronchi there is a marked increase of the connective tissue, and the tunica intima of the small arteries is thickened. The alveolar epithelium may show desquamative changes, and brown and yellow pigment granules may be present.

Interstitial pneumonia is often found in association with congenital syphilitic lesions in the skin, with interstitial hepatitis, and with changes in the epiphyses; but it is also found in cases in which gummata are present in the lungs, liver and other organs. The change begins during fœtal life, and at birth may have affected the lungs extensively. In such cases life is of short duration and death occurs from asphyxia, as is shown after death by the frequent presence of ecchymoses in the pleura, pericardium and thymus gland. When the change is less advanced at birth, such children may die at a later period by a slow process of carbonic acid poisoning, the first sign of which may be that a child previously fretful and noisy becomes quiet.

In cases in which the other organs are healthy, or nearly so, life may be prolonged for months or years; such subjects are, however, specially liable to acute disease of the respiratory organs, such as pleurisy, acute bronchitis, and broncho-pneumonia.

From the above description it will be seen that the morbid processes concerned in the production of the gummatous and diffuse changes found in the lungs of syphilitic children chiefly affect the connective tissue and small arteries. They are—(i.) A round-celled infiltration and proliferation of the interlobular and interalveolar connective tissue, originating in the cellular tissue around the bronchi, and leading to marked thickening of the framework of the lung. (ii.) An isolated perivascular cell proliferation, which begins around the small arteries, and is accompanied by changes in the tunica intima (Hochsinger). Both the periarteritic and peribronchial granulations may occur as separate nodules or node-like foci; or they may be diffused over large portions of the lungs. A well-marked desquamation of the alveolar and bronchial epithelium is almost always present, but it is quite a secondary process.

In the account here given of the pulmonary changes found in hereditary syphilis the descriptions of Heller, Spaundis, and Hochsinger have been followed, and to these authors I desire to acknowledge my indebtedness.

Association of congenital syphilis and pulmonary tuberculosis.—Syphilis, by lowering the resisting power of the individual, may dispose to tuberculosis; and it has recently been shown by Hochsinger that the virus of syphilis and tuberculosis may be jointly transmitted from parent to offspring.

This observation is of much importance, and throws a new light upon the nature of the pulmonary lesions found in infants the subjects of congenital syphilis. Hitherto it has often been assumed, on evidence which is now proved to be insufficient, that such lesions are of syphilitic origin; whereas it is clear that they may be due to an associated tuberculous infection.

In three infants suffering from congenital syphilis, and presenting symptoms of infiltration of the lungs, the pulmonary disease was found after death to be due to tuberculosis and not to syphilis. Tubercle bacilli were found in the lungs in all the cases.

The first case was observed in 1891 in a child not quite three weeks old; the second in 1891 in a child twenty-four days old; the third in 1893 in a child eleven weeks old.

Case I.—Anna B., æt. nearly three weeks. The parents had been married nine years. The father acquired syphilis shortly before marriage. The mother died from pulmonary tuberculosis three months after the birth of the child. The first and second children of the marriage were still-born; the third and fourth died during the first week; the fifth and sixth were living, ages four years and two years respectively. The case of the seventh child is here described. From the time of birth she was sickly and suffered from nasal obstruction, snuffles, and dyspnœa. Râles were present in the chest. At the end of the second week a bullous eruption appeared on the nates. The child presented all the ordinary external signs of congenital syphilis, and was shown at the Vienna Dermatological Society as a case of gummatous disease of the viscera.

On examination of the chest there was marked dulness on the left side from the angle of the scapula downwards, with bronchial breathing over the dull area. The respiratory murmur was harsh over both lungs, with rhonchi and coarse râles. The spleen was enormously enlarged, extending as low as the anterior superior spine of the ilium; the liver could be felt four fingers' breadth below the costal margin, it was hard and the edge was rounded. The diagnosis was pulmonary and visceral syphilis. Mercurial treatment was prescribed. The child died on the thirty-first day after birth. On post-mortem examination the internal organs were found extensively infiltrated with tubercle. Both lungs showed tubercles varying in size from a miliary granulation to a walnut. A nodule as large as a hen's egg occupied the right middle lobe. The left lobe of the liver was almost completely replaced by a caseous nodule; numerous tubercles studded the right lobe. The spleen was enlarged to nearly four times its normal size and contained similar deposits. Tubercles were also present in the kidney, pericardium and peritoneum. The mesenteric and bronchial glands were enormously enlarged, and in many places caseous. Tubercle bacilli were present in all the lesions. None of the lesions in the internal organs was of syphilitic origin.

Case II.—Victoria S., twenty-four days old. The mother had previously brought three children suffering from congenital syphilis to the same clinic. She had previously stated that she had not had syphilis.

Nothing was known of the father, and it is not certain that either parent was tuberculous. The child had snuffles and presented all the characteristic appearances of congenital syphilis. There was a confluent papular syphilitic eruption on the nates and elsewhere. The percussion note over the left lung was dull and the breathing bronchial, with consonating râles. The spleen was slightly, and the liver markedly, enlarged. The temperature was normal. Mercurial treatment was ordered.

The patient was shown at the Vienna Dermatological Society as a case of syphilitic pemphigus and syphilitic pneumonia. The child died on the thirty-eighth day.

On post-mortem examination the left lower lobe was solid from grayish white infiltration. There was acute catarrh of the bronchi of the left upper lobe and throughout the right lung, also of the larynx and trachea. The mediastinal and bronchial glands were enlarged, but not obviously caseous. The liver was large, reddish brown, somewhat indurated and with rounded margin. On microscopical examination of the lungs confluent peribronchial and perivascular tuberculosis was found, with tubercle bacilli. In the liver recent interstitial inflammation was present, with fatty degeneration of the liver-cells. No trace of tuberculous lesions were found in any other organ than the lungs.

Case III.—Auguste G., eleven weeks old. The mother, æt. 28, was suffering from pulmonary tuberculosis. She had had five illegitimate children and denied having had syphilis. Nothing was known of the father. The child presented the characteristic appearances of congenital syphilis, and had snuffles and a syphilitic rash on the buttocks. The rash appeared during the third week. The child had suffered from cough since it was five weeks old. There was doubtful dulness over the right lower lobe with bronchial breathing and abundant moist râles. The liver was very large and hard, with a rounded edge. The spleen extended four fingers' breadth below the costal margin. The temperature was normal. Mercurial inunction was ordered. The child died aged sixteen weeks.

Post-mortem.—The right lower lobe was solid from a homogeneous, grayish white infiltration. Grayish red and yellow tubercles were disseminated throughout the upper lobe. The lower half of the left lower lobe was collapsed. The bronchial glands were enlarged and caseation was beginning. The liver was fatty and slightly granular. In the portal fissure there was a caseous lymphatic gland the size of a hazel-nut. The mesenteric glands were caseous. The spleen contained a large caseous nodule. Microscopic examination showed the characteristic signs of "chronic tuberculous broncho-pneumonia, tuberculosis of the spleen and mesenteric glands, and syphilitic interstitial inflammation of the liver with well-developed inflammation of the vessels."

It is clear from the perusal of these cases that it will be necessary in future, even when the evidence of syphilis in the fœtus is undoubted, to examine carefully for tubercle bacilli before a pulmonary lesion is attributed to syphilis.

Acquired syphilis.—To present a trustworthy account of the morbid anatomy of acquired syphilis of the lungs is a far more difficult task than that just attempted. In considering a matter of such uncertainty I have preferred to rely upon evidence which is at hand and may be put to the test, rather than upon that to be found in the records of a period when, owing to the absence of any certain test for tuberculosis, the difficulty of distinguishing between the pulmonary lesions of tubercle and syphilis was almost insuperable.

A study of the specimens of pulmonary syphilis contained in the London museums shows that the possibility of the changes being due to tubercle was in nearly all cases carefully considered. These specimens and the records connected with them probably constitute the most trustworthy evidence on which to base a description of the morbid anatomy of the acquired disease, and, as will be seen hereafter, they have been fully utilised.

Pathology and Morbid anatomy.—Bronchial catarrh may occur as a manifestation of the secondary stage of syphilis, and possibly also of the period of incubation (Walshe). The fact that bronchitis, occurring without obvious cause in syphilitic subjects, may be greatly alleviated or cured by the administration of mercury, is strongly in favour of this view. In the late secondary and tertiary stages gummatous infiltration of the submucous tissue of the trachea and bronchi is not infrequent, and may be followed by the formation of fibrous tissue which, subsequently undergoing cicatrisation, produces stenosis, one of the most characteristic syphilitic lesions in the main bronchi.

No definite statement can be made as to the most common period of the occurrence of gumma in the lungs; cases of which the real nature could not be doubted have been recorded as early as two years and as late as twenty years after infection.

The pulmonary lesions of acquired syphilis belong chiefly to the late tertiary stage of that disease.

A. *Gumma.*—Gummata may occur either singly or in numbers, and may vary in size from that of a hemp-seed or a hazel-nut to that of a hen's egg, but the latter size is of rare occurrence. A gumma may be found in any part of the lung, but more commonly within its substance than upon the surface; and more often about the root, near the large vessels and bronchi, than elsewhere. The lower lobes are perhaps more often affected than the upper.

A gumma is rarely seen in the very early stage, of which alone the name is in any sense descriptive; but it is said then to present a gelatinous or glutinous appearance (1), thus resembling a similar growth in the liver. At a later stage it is of a gray colour, tinged with various shades of a red, white, or yellow, and presents on section a smooth and semi-transparent appearance. At a still later period a gumma forms a well-defined nodule of a yellowish colour, firm and dry. Inflammatory changes in the surrounding lung may lead to the production of a well-marked fibrous capsule, but this may be absent. The gumma may break down, and, its contents having been discharged, an irregular cavity may result; but this is, both absolutely and also in comparison with the occurrence of a similar change in caseous tuberculous masses, very rare.

The chief difference between a gumma of the skin, for example, and one of the lung is that whilst the former tends towards necrosis the latter tends to be transformed into a mass of scar tissue, the contraction of which causes puckering of the surrounding lung and overlying pleura. By the deposition of lime salts a gumma may become calcareous.

In histological structure a gumma of the lung does not differ essentially from a similar growth elsewhere. In the early stage it is seen to consist of a granulation tissue composed of small cells about $\frac{1}{2500}$ in. in diameter, arranged concentrically around the sheath of the small vessels, and in some cases around the small bronchi. At a later stage the nodule becomes opaque in the centre, and its cellular structure can no longer be recognised; but fatty and albuminous granules are seen instead in the meshes of a dense fibrous stroma. Finally it becomes converted into a mass of dense cicatricial tissue. A gumma may form a centre from which a small-celled growth may infiltrate the surrounding tissue, spreading chiefly along the bronchioles.

The walls of the neighbouring alveoli are also infiltrated with small cells; and the alveolar spaces contain inflammatory products, due either to epithelial proliferation or to the presence of cells of a character similar to those constituting the nodular masses already described. Giant cells are occasionally present, but are not so characteristic a feature of gumma as of tubercle.

According to Dr. Councilman, the essential process in the production of a gumma in the lung is a pneumonia with fibrinous exudation, accompanied by fibrous change in the alveolar walls, the whole subsequently undergoing caseation. The first step in the process is stated to be a hyaline degeneration of the capillaries of the affected area; this is followed by atrophy of the alveolar walls. The alveoli become distended with large pale epithelial cells and fibrin; the cells also undergo the hyaline degeneration, forming smooth bodies staining with eosin, and varying in size from one-half the diameter of a red blood corpuscle up to that of a large epithelial cell. The capillaries become converted into rigid tubes and their lumen is much narrowed. Similar changes occur in the small veins and arteries. Immediately around the bronchi and arteries there is a formation of connective tissue, and here the alveolar walls show much thickening and contain many small round cells.

The whole of the structures thus altered tend to undergo necrosis, and when that change is complete a caseous-looking mass results.

The following descriptions of specimens in the Museum of Guy's Hospital well illustrate the appearances presented by gummata in the lungs. It will be observed that all the specimens here described were removed from the lungs of adults.

No. 254.—A portion of the upper lobe of a lung showing on the cut surface two masses, one of which was described in the recent state as "consisting of a circumscribed nodule of a firm, yellowish, dry substance, corresponding in all particulars to that in the liver (a gumma), except in being somewhat less firm: the other is softening, breaking up, and in process of forming a cavity. Histologically the nodules are seen to consist of fibroid tissue with many areas of caseation and a few giant cells.

From the report of this case by Dr. Wilks the following additional particulars have been derived:—The patient was a sailor, aged 29. No history was obtained; he was moribund from laryngeal obstruction when admitted, and there was profuse expectoration of mucus and blood. There was a scar in the groin, and phimosis from a contracting sore on the penis. The whole mucous membrane of the larynx and trachea was deeply ulcerated, and the walls thickened by an infiltration of fibrous tissue into the submucous structure, producing great induration. The thyroid cartilage was bare at one spot, the lymphatic glands in the neck were enlarged.

The liver contained a dozen hard, round, fibrous tumours—the largest the size of a marble—yellowish white, tough, and of leathery consistence, dry, and emitting no juice on pressure. In two or three the circumference of the tumour consisted of a translucent structure; and this was evidently the more recent formation, the opaque and yellow parts being probably tissue undergoing a degenerative change. At one spot a deep cicatricial appearance was produced by the contraction of a group of these small nodules.

Microscopically the nodules consisted of nucleated fibres and fibrous tissue.

No. 255.—The lower lobe of a left lung from a man, aged 27, who died from erysipelas of the larynx. The specimen shows at its hinder part a large yellowish mass partially separated from the surrounding tissue. Smaller nodules are seen in the adjacent lung. The pleura over the gumma is much thickened. Histologically the nodule consists of fibrous tissue which stains with difficulty. There were many gummata in the liver. With the exception of the above lesions and some bronchitis the lungs were healthy. There was a chancre on the penis and suppurating buboes.

No. 256.—A portion of lung showing scattered through it several small masses of irregular shape, yellowish in colour, and firm on section. These masses are easily separable from the surrounding lung, which is healthy. Histologically the nodules show a central area of caseous material surrounded by a narrow zone of fibrous tissue in which are many small round cells. The liver contained a single gumma, and was in a condition of diffuse syphilitic hepatitis. There were several gummata in the testes. From a man, aged 39, who had suffered from cough and dyspnœa for six months. He was admitted for hepatic ascites and slight jaundice.

The following specimen from the same Museum illustrates the appearances presented by a gumma which has undergone fibrous transformation:—

No. 253.—A section of a right lung. From a man, aged 36, admitted for fracture of the cervical spine. At the upper part of the lower lobe is a circumscribed patch of fibroid material with radiating processes extending into the surrounding pulmonary tissue. The pleura over it is much thickened. The interlobar septum is thickened, and from its upper portion similar fine fibrous strands radiate into the upper lobe. Other portions are very emphysematous (also fibroid and pigmented). No tubercle was found anywhere. There was lardaceous disease of the liver, spleen, and kidneys. Both testes were good specimens of syphilitic orchitis.

The following description of a specimen in the Museum of St. George's Hospital illustrates a combination of the caseous and fibrous stages of a gumma (10):—

"Section of a right lung near the root. In the posterior and upper part of the lower lobe, close to the spine, there is an area showing marked fibrosis; situated within it is a caseous mass the size of a marble, somewhat loose. The overlying pleura is adherent and thickened; bands of thick grayish fibrous tissue pass inwards from the pleura, and joining with each other form a meshwork." No tubercle in any organ; surface of the liver scarred from perihepatitis. Large caseous gumma near the portal fissure, with smaller ones in its neighbourhood. Liver cirrhotic and lardaceous. Gummata in both testes. From a male patient who contracted syphilis in 1884, six years previous to his death. In 1886 he suffered from syphilitic disease of the testes and sores on the right elbow. Death was due to uræmia.

Lobular or Broncho-pneumonia.—A careful review of the evidence on which it is believed that inflammatory changes of the lobular or bronchopneumonic type occur as the direct result of syphilis impresses me with the conviction that many of the cases described in the past as presenting such lesions were really cases of tuberculosis.

In the following case (3), however, such a possibility may be excluded. It will be observed that the pulmonary lesions were secondary to and in continuity with the growth of large gummata in the liver and spleen. The specimen is in St. George's Hospital Museum :—

Left lung.—The lower lobe is deeply congested and partially consolidated; the consolidation is in patches as in catarrhal pneumonia. Some of these masses appeared purulent, others fatty or caseous. The size varied from 3 mm. to ½ mm.; each patch or nodule was surrounded by a deeply congested zone. Right lung.—The lower lobe presented changes similar to the above; it was adherent to the diaphragm, through which a large caseous gumma in the liver had extended into the lung. At the upper margin of the caseous mass there was much fibrous induration and exudative consolidation of the pulmonary tissue. For the microscopical changes, which are given in great detail, the reader is referred to the original article. There was a gummatous mass chiefly in the upper part of the right lobe of the liver measuring 5½ in. by 4¼ in., and another occupying the upper third of the spleen. That organ was greatly enlarged, weighing 2 lb. 6 oz. Both liver and spleen were firmly adherent to the diaphragm, and the muscular tissue of the latter was in part destroyed by the extension through it of the gumma in the liver. The specimen was taken from a man, aged 43, who contracted syphilis in 1861, twenty-five years before his death. He had periostitis of the tibia in 1864, left hemiplegia in 1871, and again in 1876.

Fibroid induration.—The following are the more important changes of this nature which have been attributed to syphilis: (*a*) thickening extending from the hilum around the bronchi and vessels; (*b*) isolated masses of fibroid tissue in various parts of the lung; (*c*) diffuse changes occupying the whole or the greater part of one lung.

The marked tendency of gummatous lesions to spread along the vessels and bronchi has already been referred to.

The following case (4) is an example of syphilitic fibrosis illustrating the first variety of this lesion:—

Woman aged 50.—Thrombosis of cerebral artery; hemiplegia. Pigmented excavated scars on left leg, due to old syphilitic ulceration. Lungs.—Emphysema. Right lower lobe contained a deep depression and a much-puckered cicatrix due to pigmented fibroid bands running into the lung tissue. No caseous or calcareous nodules. No pleural adhesions. Microscopical examination.—The fibroid tissue is arranged chiefly around the vessels and bronchi with a more or less concentric disposition. The coats of the vessels are much thickened. There is a small-celled growth invading the alveolar walls, which are also much thickened. In places the cells and nuclei are aggregated in heaps.

As an example of fibrosis in the form of scattered areas of induration, the following case may be cited from the same source (4):—

Woman aged 25.—Fracture of cervical spine. Pigmented and puckered cicatrix and syphilitic ulcers on left leg. Calcified gumma in the liver. Right lung.—Upper lobe healthy. Middle lobe presented in the centre large irregular patches formed by radiating bands of fibroid tissue; also smaller scattered patches of the same nature: the bands whitish, not pigmented. One patch contains a calcified nodule. No pleural adhesions. Left lung.—Adhesions over lower lobe; and whitish, puckered, depressed fibroid patches with irregular thickening of the pleura. On section extensive fibroid infiltration; bands appear to run into the lungs from the pleura. Some small rounded caseous patches are also present.

The following specimen from the Museum of Guy's Hospital (9) illustrates the appearances met with in "diffuse syphilitic fibrosis of the lungs." The patient was a man, æt. 54, who had suffered from winter cough for some years.

No. 252.—A portion of a right lung in which there is a considerable excess of fibroid material appearing on the cut surface as a delicate network traversing the pulmonary tissue in all directions. The fibroid change is less marked at the apex than at the base, in which latter situation many of the air-vesicles are dilated; over this area the pleura is slightly thickened and is adherent. The dense fibroid tissue that pervades the lung shows, scattered through it, numerous collections of small round cells not undergoing caseation. No giant cells are present. The walls of the small arteries are thickened. One or two small cavities the size of peas, with soft caseous contents, were situated near the root of the right lung, probably softening gummata; no tubercle bacilli could be found in them. The condition of the left lung resembled that of the right. The liver was scarred; the testes were fibroid. Death was due to bronchitis.

Changes in the bronchial glands and lymphatics of the lung.—In a case of syphilitic disease of the liver, lungs, dura mater, cranium and sternum, recorded by Dr. Hermann Weber, the bronchial glands and lymphatics of the lung presented the following appearances:—The bronchial glands were much enlarged—some being of the size of a pigeon's egg, some only that of a hazel-nut. From the grayish white section of the larger glands, which were rather soft, a creamy fluid exuded, consisting of fat globules, granular corpuscles, and an abundance of large cells in a condition of fatty degeneration. The less enlarged glands were harder, their sections offered a marbled appearance, large white patches, almost like bacon, being interspersed with grayish red, very vascular tissue. No juice exuded spontaneously or could be squeezed from the section. Large nuclei and nucleated cells were the principal microscopical elements, with a very small proportion of fibres thickly studded with nuclei. The lymphatics leading from the lungs to the enlarged glands were dilated and their ramifications on the surface and throughout the lungs were distended with creamy fluid.

A similar appearance is described in the case of Drs. Delépine and Sisley already quoted. "Immediately under the pleura there was a network composed of ramified tracks. The appearance suggested lymphatics distended with cells or some fatty products." The lymphatics of the subserous layer of the pleura were considerably enlarged over areas corresponding to the yellow patches (? of syphilitic bronchopneumonia) within the lung.

Dr. Weber rejects the view that the bronchial glands were first affected by the syphilitic virus, and that the engorgement of the pulmonary lymphatics resulted from obstruction to the passage of the lymph.

A progressive destructive disease, the so-called "syphilitic phthisis."

It appears to me that the question of the existence of a syphilitic lesion of the above form can only be settled by a careful study of cases which fulfil the following conditions:—

(i.) The cases must be complete; that is, the symptoms observed during life must be considered in connection with the lesions discovered on post-mortem examination.

(ii.) The evidence of syphilitic infection must be undoubted.

(iii.) Repeated examinations of the sputum must have been made, and tubercle bacilli invariably absent; and the absence of tubercle from the lungs (as the cause of the lesions) must be proved by post-mortem examination.

(iv.) Syphilitic lesions about the nature of which there can be no doubt must be found in other organs.

From such evidence alone can we hope to construct the clinical history and morbid anatomy of advanced syphilitic disease of the lungs.

The following cases illustrate this variety of the disease :—

Case I.—Charles N., æt. 38, bricklayer. In 1892 he suffered from cough, with expectoration and pain on the left side of the chest. In 1893 he had night-sweats and dyspnœa. From January to May 1894 he was an in-patient of the Brompton Hospital under the care of Dr. Mitchell Bruce; the diagnosis then recorded was "Syphilis (?), tracheal stenosis, chronic bronchial catarrh, induration of the left upper lobe and of the left base with pleural adhesions over that area. Cicatrisation of the soft palate and adhesions of the right posterior pillar of the fauces to the back of the pharynx." There were no bacilli in the sputum. He continued fairly well until October 1894, when he expectorated a large quantity of offensive purulent material for two consecutive days. Cough was very severe at this period. His health subsequently improved, and so remained until 20th February 1895; when in the course of a few days he brought up about a quart of blood-stained sputum. Hæmorrhage then ceased and dyspnœa diminished. On 5th March, cough and dyspnœa increased and he became seriously ill, with constant headache and slight delirium. Œdema of the feet subsequently supervened. On 15th March 1895, he was admitted to the Brompton Hospital under the care of Dr. Percy Kidd. On admission he was reported to be fairly well nourished. He stated that he had not lost weight, and, beyond an occasional streak of blood in the sputum, there has been no hæmoptysis. There was marked stridor and severe cough. Right lung resonant everywhere; breath-sounds much exaggerated, expiration prolonged. Loud hoarse inspiratory and expiratory stridor all over the lung; sibilant rhonchi general. Left lung.—Expansion much diminished; resonance much impaired front and back; breath-sounds weak; expiration prolonged; fine crackling rales over the whole of lung; vocal fremitus and resonance diminished. Expectoration profuse and difficult to expel. No tubercle bacilli found. Temperature, 99° F. It varied between that point and 96° F. during the time the patient was in hospital. The dyspnœa gradually increased, and death occurred on 10th April.

Necropsy.—Scars on tongue, glans penis, and scrotum; and adhesions of skin to left testis. Marked thickening of the right tibia. Larynx normal. Trachea narrowed at the lower end. Recent ulceration from cricoid downwards for two inches; below this, down to point of bifurcation, there was extensive scarring of the cartilaginous portion; and also at its line of junction with the posterior wall. The submucous tissue was extremely thickened. Cartilages bare in several places. The main bronchi were much scarred and showed extreme narrowing. The bronchus to the left upper lobe was impermeable to a probe. Right lung.—Old pleural adhesions over the upper lobe, recent pleurisy with effusion at the base. Emphysema, with reticular fibrosis especially around bronchioles. Deep in the upper lobe at the edge of one of the main bronchi there was a large black fibroid mass, with fibroid radiation into the surrounding tissue; elsewhere two small, hard, raised masses, one with fibrous strands running up to it. Base solid from bronchopneumonia. No appearance of tubercle. Left lung.—Upper lobe extremely contracted, containing no normal tissue. It consisted of deeply pigmented blackish gray fibrous tissue surrounding the openings of bronchial tubes, and bronchiectasis. At the centre there was a smooth-walled cavity about the size of a small chestnut into which a bronchus opened. No appearance of tubercle. Lower lobe.—Emphysematous, with reticular fibrosis along the margin and at the base. Bronchi dilated, but not to a marked degree. About the centre point of the outer margin there was a small nodule, probably a gumma; white and firm, and surrounded by a pigmented fibrous capsule. The extreme base consisted of indurated fibrous tissue extending from the pleura to a cavity, the size of a marble, into which a small bronchus opened. From this cavity fine fibrous bands radiated in all directions, producing extensive fibrosis of the surrounding lung. Perihepatic and splenic adhesions. Liver scarred and nutmeg. Spleen contained several calcareous masses surrounded by a fibrous capsule. Testes fibrous.

Case II.—R. D., æt. 36; coachman. The family history is unimportant. At the age of 18 he had a sore on the penis, for which he was treated for several months with medicine and a lotion. In 1890 he became an out-patient under the care of the writer at the Brompton Hospital; he was suffering from cough and expectoration, which continued. There was an enlarged gland in the inferior triangle of the neck on the right side, dulness at the right apex, with feeble breath-sounds, and bronchial breathing in the right supraspinous fossa. The liver was large, nodular, and very tender. He took iodide of potassium in gradually increasing doses and obtained some relief. He was subsequently an out-patient at the Middlesex Hospital. In March 1893 he caught a severe cold, but remained at work. In following April he noticed œdema of the legs and scrotum. He was admitted into Middlesex Hospital, under Dr. Cayley, on 13th May 1893. He was pale and emaciated, the legs and scrotum were œdematous. He had troublesome cough, accompanied by the expectoration of large quantities of extremely fœtid pus. The breath was fœtid.

Physical signs.—Expansion deficient on right side. Relative dulness at right apex front and back, breath-sounds feeble over dull area. Absolute dulness from level of fifth interspace in nipple line and in axilla to base; behind from angle of scapula to base. Vocal fremitus and resonance diminished, and breath-sounds scarcely audible over dull area. Left side normal. No displacement of heart. Hepatic region prominent. Liver dulness extended 3 inches below the costal arch in right mammary line and almost to umbilicus in middle line. Liver somewhat soft and elastic. Urine, sp. gr. 1004, neutral, contained albumin and fatty casts. The expectoration consisted of frothy greenish pus, forming thick masses in a watery fluid. It contained no tubercle bacilli.

17th May.—The chest was explored in the axillary and submammary region. No pus was found. 19th May.—Liver exposed by incision below costal arch, and a depressed cicatrix seen. The expectoration continued copious, green, and fœtid. Absolute dulness appeared over whole of right side up to clavicle, with amphoric breathing and pectoriloquy below clavicle. 15th June.—Offensive pus was evacuated through a canula inserted in third right interspace in mid-axillary line: a portion of the fourth rib resected, lung incised, more pus evacuated, drainage-tube inserted. 19th and 20th June.—Hæmorrhage from wound. 21st June.—Death.

Necropsy. Abstract of notes:—Old syphilitic scar in trachea, six rings above bifurcation, more recent scar at bifurcation, producing stenosis of the main bronchi to the right upper and lower lobes. One bronchial gland enlarged. Pleura over right lower lobe adherent and much thickened. Bronchi much dilated beyond the site of stenosis. At the base of the upper lobe were two large irregular cavities with sinuous outlines communicating with large bronchi, lined by a distinct membrane, and containing sloughy portions of lung tissue. The anterior cavity had been opened by the incision. The section of the lung was smooth and presented a finely speckled yellow appearance. No pus exuded from the yellow spots on pressure. In the anterior part of the lower lobe there was a large irregular cavity, the walls of which showed no sign of any mucous membrane; they were covered with yellowish gray sloughy material. No tubercle present and no caseation. The lung puckered in many places and fibrous almost throughout. Liver enlarged (76 oz.). Large puckered cicatrix on the upper surface of the left lobe and many similar cicatrices elsewhere. A cretaceous and caseous gumma on the posterior aspect of the right lobe. Liver substance fatty and amyloid. Kidneys large, pale, lardaceous, and fatty.

Case III.—T. H., æt. 59; painter. Admitted into Middlesex Hospital under Dr. Fowler, 4th February 1893. Father died aged 70; mother aged 75. No history of tuberculosis in family. Accident to left knee æt. 19 years, followed by formation of an ulcer. Chancre on penis in 1858 (æt. 25), secondary rash and sore throat subsequently. In 1864 ulcers on left leg and twice subsequently. In 1880 ulcer on right leg, near external malleolus. Dry cough since 1887, worse in winter. Since December 1892 severe paroxysmal cough with offensive muco-purulent expectoration. Marked emaciation during this period.

A pale, gray-haired, emaciated man. Breath very fœtid. Extensive scars on left leg of old standing, more recent scars on right leg. Scar in right lumbar region where incision was made for "abscess." Right lung.—Hyper-resonant on percussion; breath-sounds at apex bronchial, front and back; crackling rales in supraspinous fossa. Bronchophony and pectoriloquy well marked in same area. Dulness over lower lobe to angle of scapula, breath-sounds bronchial, with coarse crackling rales over same area. Left lung.—Resonance impaired over clavicle and in supraclavicular fossa, elsewhere hyper-resonance. Bronchial breathing over upper lobe, front and back, with crackling rales. Breath-sounds bronchial over upper part of lower lobe, with bubbling and coarse crackling rales, the latter extending to the base. Urine, sp. gr. 1020; no albumin. Expectoration copious, purulent, and offensive. Frequent examinations made for tubercle bacilli, but none found. No elastic tissue found. Temp. 98°, pulse 84, respirations 44. 21st February.—Dulness at both apices, and medium crackling rales. Temperature between 99° and 100° F. The respirations between 36 and 48. Severe cough, and the breath and expectoration offensive. Died 23rd February.

Abstract of P.M. notes:—Scar on corona of penis with some induration around. Calvarium thickened, dura mater adherent. Pleural adhesions over both lungs. Right lung.—Emphysema along anterior margin and at base. Apex pigmented and consolidated from pneumonia and œdema. In lower part an oval cavity measuring 2½ inches by 2 inches, in communication with main bronchus, and containing greenish yellow, offensive, shreddy material. Below this for 1½ inches the lung gray in colour and almost solid, a few small cavities with curdy contents. No tubercle found. The pleura covering the consolidated area much thickened. Left lung.—Upper lobe pigmented and "nodular." A cavity, from bronchial dilatation, occupies the posterior portion. The lower lobe emphysematous, and contained numerous encapsuled caseous masses about 2 mm. in diameter. Bronchial glands pigmented, but not caseated. No ulceration in air-passages. No gummata in liver or spleen. Testes scarred and fibrous. Small white fibrous nodule in right kidney.

The following cases are incomplete, and do not attain to the standard of evidence laid down, inasmuch as the patients are believed to be still living:—

Case IV.—Mary G., æt. 33, married. Three children alive, three dead,—one still-born, one died a few hours after birth. Has had four miscarriages. Admitted into the Brompton Hospital, 13th June 1894, under Dr. Fowler. No history of tuberculous disease in the family. Ten years ago had some affection of the liver. Three years ago had an attack of influenza followed by pleurisy (R) and congestion of the lungs. Right pleurisy recurred in August 1893. Has had a slight cough for three years, worse since September 1893. Expectoration has been profuse, and for the last two months fœtid and of a bitter taste. In October 1893 it was tinged with blood for three weeks. Dyspnœa worse since September 1893. Catamenia ceased since the birth of the last child on 30th September 1893, at which time she caught a chill. In February 1893 patient noticed a swelling in the left loin, which at first gradually increased in size and subsequently diminished. It is slightly movable and is not tender. It is about equal in size to a small Tangerine orange, is situated rather superficially, and over the erector spinæ muscle; whether actually within the muscle cannot be determined. Emaciation, cough, and weakness have been increasing lately, and night-sweats have been continuous.

Physical signs.—Right lung.—Marked flattening of the whole of the right side, particularly in front. Measurement at right nipple level:

right 14½ inches, left 16 inches. Dulness over upper lobe, with distant cavernous breathing and bronchophony front and back. Impaired resonance over upper part of lower lobe posteriorly, where crackling rales are audible; similar rales at the right base where percussion note is dull. Left lung.—Harsh breathing general (? compensatory), no adventitious sounds. Liver much enlarged and nodular on the surface; margin irregular, extends from the fourth space to below the umbilicus. Spleen not enlarged. Urine free from albumin. Expectoration profuse and fœtid. No tubercle bacilli. From June to September the expectoration was usually fœtid. Bacilli repeatedly sought for, but never found. The cavity at the right apex extended. 5th September.—Retraction more marked at right apex. Cavity dry. Numerous crackling rales in axilla, and all over base. General improvement. Liver appears more nodular. October.—Large crackling rales over base and in axilla. Cavity at apex dry. No bacilli to be found. Discharged 13th October. Intra-tracheal injections of menthol appeared at first to have an effect in diminishing and then removing the odour of the expectoration; but subsequently the fœtor returned and appeared to be uninfluenced by their continued use. The quantity of expectoration was small during the period over which their administration extended; but it had been steadily diminishing up to the time when this treatment was commenced. The patient considered that she derived benefit from the injections. The nature of the tumour in the back was doubtful, it was believed to be a gumma in the superficial part of the muscle. Inunction of mercurial ointment was made daily into the back from 22nd September onwards.

Case V.—Edward C., æt. 47; a waiter. Admitted into St. George's Hospital, 13th April 1894, under Dr. Whipham.[1] His father and mother died of "consumption." Thirty years ago he had a hard chancre. He has had syphilitic psoriasis of the palms. He has not had hæmoptysis, night-sweats, or emaciation. A fortnight before admission he was attacked with severe pain on the right side of the chest and dyspnœa. On admission he was anæmic, and complained of cough and profuse expectoration. The skin was of a brownish tint and presented numerous old rupial scars. Right lung.—Impaired resonance over upper lobe with feeble breathing. Just below the second rib there is a small area of increased dulness and cavernous breathing with whispering pectoriloquy. There are rhonchi all over the right lung and to a smaller extent over the left. The sputum is profuse and muco-purulent. No tubercle bacilli were found on any occasion; the examinations were made by several observers. 15th April.—Ordered Potassii iodide gr. v., Liq. hydrarg. perchlor. ʒj. ter die. 28th April.—Expectoration and cough less. Physical signs at right apex less marked. 2nd May.—Discharged to Convalescent Home.

The following case illustrates the fact, first pointed out by Dr. Pearson Irvine, that stenosis of a main bronchus may give rise to destructive changes in the lung. It will be observed that the case was one in which a recent tuberculosis supervened on old syphilitic disease:—

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[1] The writer is indebted to Dr. Whipham for his kind permission to use the notes of this case.

Case VI.—Margaret S., æt. 25. Admitted into the Brompton Hospital, 25th June 1884, under Dr. Reginald Thompson. Family history good. Good health up to two years ago, when after marriage she had "ulcerated legs." No sore throat or skin eruption. Cough, expectoration, pain in left side, dyspnœa, night-sweats and emaciation have been present for eight months. On admission the fingers were clubbed; there was a large circular ulcer on the back of the left thigh with some scarring, and coppery staining about the knee and leg on the same side. Cough more or less paroxysmal; expectoration copious, nummular, and purulent. No tubercle bacilli. Right chest 15½ inches, left 16½. Dulness over left lung, absolute at base, where vocal fremitus is absent; elsewhere it is diminished. Bronchial breathing, pectoriloquy, and crepitation over left upper lobe. Breath-sounds absent at base, some rhonchus there. Slight crepitation at right base.

The ulcer on the thigh yielded to antisyphilitic treatment. The physical signs remained much the same, except that the breath-sound at the left apex became cavernous. There was well-marked pyrexia throughout. The expectoration remained copious, at times it averaged a pint in the twenty-four hours. Death occurred on 1st March 1885, and was preceded by anasarca, ascites, and profuse diarrhœa.

Necropsy.—A few small scars in the subglottic portion of the larynx. The lower half of the trachea marked by numerous stellate puckered cicatrices, involving both membranous and cartilaginous portions, but especially the latter. The origin of the left bronchus represented by a small opening just admitting a probe; the surrounding parts of the tracheal wall extremely fibrous and puckered. Slight scarring in the right bronchus about the origin of the upper lobar branch. Left lung excavated from apex to base. Numerous trabeculated cavities in the upper lobe intersected by tough pigmented bands: walls thin and smooth. The cavities larger behind than in front, in the latter region they were more numerous; and the intervening fibroid induration was more pronounced. Some bronchi appeared to expand uninterruptedly into the smaller cavities. Numerous small cavities in the lower lobe situated in indurated fibroid lung. The cavities contained extremely fœtid reddish fluid secretion, and in some places some soft putty-like material. No tuberculous nodules in this lung. The contents of the pulmonary cavities, including the liquid and caseous parts, were carefully examined for tubercle bacilli, but none could be found. Right lung crepitant, but studded with large tuberculous groups which were most plentiful in the middle lobe and lower part of the lower lobe. Lardaceous disease of thyroid, mesenteric and mediastinal glands, also of the kidneys, liver and spleen, and mucous membrane throughout the body.

The recent tuberculosis of the right lung was obviously quite unconnected with the disease in the left, which was secondary to the bronchial stenosis.

This case proves very clearly that a progressive destructive disease of the lung may result from syphilitic stenosis of a main bronchus; but it does not prove that this disintegration of the lung is due to the continued action of the specific virus of the disease, as is the case in pulmonary tuberculosis. The fact that lesions similar to those here described may occur when the narrowing of the bronchus is due to pressure from without, as by an aneurysm, shows that the bronchial obstruction is the main factor in their production. Stenosis of the bronchus is followed by retention of secretion in the tubes, and this by bronchiectasis. Decomposition of the retained secretion induces inflammatory changes in the surrounding lung, and finally the part so affected breaks down and cavities are formed.

The cases here described prove that in individuals undoubtedly the subjects of syphilis, widely-spread destructive changes may be found in the lungs; and that such lesions may occur independently of the presence of tubercle. Whether they are such as to entitle the condition to be named "syphilitic phthisis" must be decided by those who continue to use the word "phthisis," a term which many teachers have ceased to employ.

If the name "phthisis" is given to a group of symptoms and morbid changes, it can hardly be denied that a case (see Case I.) which is marked by such symptoms as severe cough, dyspnœa, emaciation, fever, night-sweats, profuse expectoration, and hæmorrhage, and which, on examination after death, is found to present signs of consolidation, fibrosis and excavation of the lungs, belongs to this category. The task before us, however, is to determine the nature of the pathological lesions of pulmonary syphilis and of the symptoms which they produce; whether they are such as to warrant the use of a vague nomenclature which it would be well to discard is a question of little importance. It may be of service, however, to draw attention to the chief points of difference between the pulmonary lesions of tuberculosis and syphilis.

I. Tubercle usually affects the apex of the lung, and subsequently the apex of the lower lobe; and tends to progress along a certain route. The primary lesion of syphilis is often about the root and central part of the lung; the disease follows no definite line of march, and gummata may be found in any position.

II. Both tubercles and gummata may undergo either necrosis and caseation, or fibrous transformation; but with caseous tubercle the tendency towards softening and cavity formation is the rule, whereas a caseous gumma very rarely breaks down.

III. The progressive destruction of the lung by a process of disintegration leading to a gradual increase in the size of a cavity, a change so commonly observed in tuberculous disease, is rarely if ever observed in syphilis, except as a secondary result of stenosis of one of the main bronchi.

IV. In nearly all cases of advanced destruction of the lung occurring in the subjects of syphilis, stenosis either of the trachea or of one of the main bronchi is present, whereas this lesion is very rare indeed in tuberculosis.

V. The cavities found in cases of pulmonary syphilis are usually bronchiectatic, but not invariably so; whereas in tuberculosis they are commonly due to progressive destruction of the lung, but may be bronchiectatic.

VI. The tendency to the formation of pulmonary aneurysms, which is so marked a feature in tuberculosis, is rarely observed in pulmonary syphilis.

VII. Pulmonary lesions in tuberculosis are very common, whereas in syphilis they are extremely rare.

The necessity for prolonged specific treatment is certainly more generally appreciated now than formerly; and it is therefore probable that rare as these lesions have been in the past, they will be still rarer in the future. The conditions which favour their development are the neglect of mercurial treatment shortly after infection, and anything which, by lowering the general health, tends to diminish the resisting power of the individual.

When our knowledge of the virus of syphilis is as complete as that we even now possess of the bacillus tuberculosis, it may be possible to state definitely whether the destructive pulmonary lesions found in advanced cases of the acquired disease are directly due to the continued action of a specific micro-organism; at present the problem remains un-unsolved.

Symptoms.—The only point worthy of mention in respect of syphilitic lesions of the bronchi is that the catarrhal signs which accompany the secondary stage are, as a rule, general in their distribution; whilst in the tertiary stage they are more often localised, owing to the tendency at that period to the formation of gummata in the main bronchi. Should stenosis occur, there may at first be bronchial breathing limited in area, and often most marked about the root of the lung posteriorly. As the lumen of the tube diminishes, the breath-sounds, over the pulmonary area which it supplies, become more and more feeble, and finally disappear when air ceases to pass the obstruction. If bronchiectasis is forming behind the site of stenosis there may be cough with profuse, purulent, and fœtid expectoration, accompanied by general signs such as emaciation and moderate pyrexia.

In the cases described in this article it will be observed that *cough* was, as a rule, the earliest and most prominent symptom. In the early stage it may be due to irritation, the result of laryngeal, tracheal, or bronchial lesions; at a later period it is probably chiefly due to the changes within the lung itself.

Dyspnœa comes next in point of frequency. It varies in severity with the nature of the lesion: slight when this is limited, in cases of extensive fibrosis or stenosis of one of the main bronchi it may be very severe. The dyspnœa tends to become paroxysmal and to assume the characters of bronchial asthma. *Hæmoptysis* has not been of frequent occurrence in cases observed by myself, but it may occur and may prove fatal. In one case of syphilis of the bronchial glands, profuse and fatal hæmorrhage occurred from softening of the gland and its rupture into a main branch of the pulmonary artery.

Expectoration may be profuse, purulent, and offensive. Fœtor of the expectoration is common in cases of advanced pulmonary syphilis. The sputum will be free from tubercle bacilli.

Pain may be present, but is not a very prominent feature of the disease.

Emaciation is not, as a rule, nearly so extreme as in tuberculosis; but with advanced lesions in the lungs the difference is not so remarkable as to be of any value from a diagnostic point of view.

Night-sweats were present in several of the cases here described.

When extensive lesions are present, *pyrexia* may be considerable, and of the hectic type commonly observed in tuberculous disease of the lungs; but in the early stages of the disease there may be a complete absence of fever.

The general symptoms, as will be seen on reference to the cases described, do not, in the presence of widely-spread lesions, differ markedly from those of advanced tubercular disease of the lung.

Physical signs and Diagnosis.—The lesions of syphilis are rarely of such a kind as to produce signs by which they can be distinguished from others of an entirely different origin.

Consolidation and excavation will be recognised by their ordinary signs, probably before their syphilitic origin is suspected; and it appears therefore unnecessary to describe them in detail, more particularly as in the cases here recorded the results of the physical examination are given in full.

The features of pulmonary syphilis are certainly not as yet so clear that the disease can be recognised by any positive signs; but by a process of exclusion a diagnosis may generally be made.

The case will probably be regarded at first as one of pulmonary tuberculosis; but repeated examination of the sputum and the failure to discover tubercle bacilli will suggest another origin.

A careful inquiry, previously perhaps omitted, will now be made as to syphilitic infection and as to the occurrence of any secondary or tertiary manifestations of this disease. The absence of such a history in a hospital patient will not exclude syphilis; but it is rare in private practice for a patient to have had syphilis with tertiary symptoms and to be ignorant of the fact.

Evidence of tertiary lesions in the larynx, liver, spleen, or testes is of importance as showing that the viscera are affected.

Careful search should also be made for lesions of the calvarium, of the dura mater, and of the sternum and ribs.

Speaking generally, the diagnosis of pulmonary syphilis from tuberculosis will depend far more upon the examination of the sputum than on the results of physical examination.

A careful examination of undoubted specimens of pulmonary syphilis does not bear out the statement that the lesions are generally limited to the middle part of the lung; they are so often found elsewhere that their more frequent occurrence in that part ceases to be a fact of much value in diagnosis. It would be rash indeed to diagnose pulmonary syphilis because of a lesion situated in and apparently limited to the middle of one lung, without having previously demonstrated, by frequent examinations, the absence of tubercle bacilli from the expectoration. Such points, however, are not without importance, as being unusual in a case possibly hitherto regarded as one of "phthisis" or "consumption," they may serve to arrest attention.

Evidence of excavation and the expectoration of a fœtid sputum, which does not contain tubercle bacilli, should always suggest the possibility of pulmonary syphilis. When the physical signs indicate stenosis of the trachea, or of one of the main bronchi, and the presence of a growth or an aneurysm can be excluded, it is very probable indeed that syphilis is the main factor in the case.

Those who are content to diagnose "phthisis," and neglect the systematic examination of the sputum, will almost certainly overlook a case of pulmonary syphilis if it should come in their way.

A striking example of this has recently come under my notice. A military officer who had contracted syphilis some years back began to suffer from symptoms of laryngitis; and on examination of the chest well-marked signs of disease were found at the apex of the right lung. The laryngoscopic appearances did not suggest to several competent observers that the lesion was due to syphilis, and the case was regarded as one of "consumption of the throat and lungs." It occurred to a medical man who saw the patient at a later period to examine the sputa for tubercle bacilli, and, as none was found on repeated examination, doubt was cast upon the diagnosis of "phthisis"; mercury and large doses of iodide of potassium were prescribed, and the patient rapidly improved; but the stenosis of the larynx remained.

Prognosis. — Extensive pulmonary lesions, particularly excavation whether of bronchiectatic or disintegrative origin, and fœtid expectoration are certainly very grave complications of syphilis. If, moreover, there is evidence also of gummatous hepatitis, albuminuria, and lardaceous disease, recovery is scarcely possible, and life is not likely to be much prolonged.

It is probable, however, that, with our present improved means of diagnosis of tuberculosis of the lungs, syphilitic cases, which formerly would have been considered tuberculous, may be recognised as syphilitic at an earlier stage, and the patients under appropriate treatment may recover. In an undoubted case of pulmonary syphilis, which came under my own care at a late stage of the disease, the affection had been kept in check for many years by repeated visits to Aix-la-Chapelle, and by the active employment of antisyphilitic treatment. In any case seen in an early stage, great improvement, if not complete cure, may reasonably be expected from the use of similar measures. There are, however, limits to the action even of specific remedies; and it is not to be expected that lesions such as bronchial stenosis and dilatation, extensive fibrosis and excavation, or gummata in a state of fibrosis will disappear under the administration of mercury or iodide of potassium.

Treatment.—If the disease in the bronchi or lungs is recognised in an early stage, the patient should be advised to undergo a prolonged course of treatment with mercury. Iodide of potassium in gradually increasing doses is generally administered at the same time.

If, however, the disease is advanced, and the patient emaciated, it is better first to try the effect of iodide of potassium alone; giving at the same time cod-liver oil and tonics. To maintain and improve the strength and general nutrition of the patient are matters of as much importance in the treatment of syphilitic as of tuberculous disease of the lungs, and are to be secured by the same means.

The warm sulphur baths of Aix-la-Chapelle, in association with mercurial inunction, enjoy a special reputation in the treatment of syphilis, and are to be recommended to sufferers from pulmonary syphilis who are able to go abroad for treatment.

When tuberculous disease of the lungs occurs in a syphilitic subject, the treatment will be mainly such as is suited to cases of tuberculosis. A mercurial course is rarely admissible, but iodine, in the form of the syrup of the iodide of iron, may be given with advantage.

In cases accompanied by fœtid expectoration, creasote vapour baths and intra-tracheal injections of guaiacol should be tried.

Cases of syphilitic disease of the lung accompanied by bronchiectasis have not, in the experience of the writer, been benefited by surgical measures undertaken with a view to drain the cavities.

J. K. Fowler.

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J. K. F.

DISEASES OF THE PLEURA

INTRAPLEURAL TENSION

In health the two layers of the pleura are in close contact, but they are subject to a constant strain, which tends to separate them; this is called the intrapleural tension. It is for all practical purposes equal to the elasticity of the lung, but opposite in direction; and thus the elasticity of the lung is positive and the intrapleural tension negative.

Whether in health there is any force existing between the layers of the pleura—such as that of cohesion, as I suggested some years ago (1), which neutralises the elasticity of the lung when fully expanded—is a matter which is open to question. It is possible, and I think it probable; but the question need not be further considered here.

The forces, of which the intrapleural tension is the resultant, are, first, the rigidity of the chest walls; secondly and chiefly, the elasticity of the lungs; and, thirdly, the movements of respiration.

So far as the condition of the chest walls is concerned, where they are fairly rigid, as in the adult, this factor may practically be disregarded; but not so in infants or little children, in whom the chest walls are soft and yielding; for then, under pathological conditions, part of the force which would otherwise tend to separate the two layers of the pleura is spent in drawing the chest walls in.

The condition of the chest walls and the elasticity of the lungs cannot vary while observations on intrapleural tension are being made; but the third factor, namely, the movements of respiration, is one which is constantly varying, and introducing variations in intrapleural tension which have to be reckoned with. Thus during inspiration the lungs are placed more on the stretch, and consequently the intrapleural tension is greater; during expiration the lungs are less on the stretch, and the intrapleural tension is therefore smaller.

If the air in the tubes were stationary, as it is after death, the pressure in the air-tubes would be that of the atmosphere; but, during respiration, the air, as it passes in and out through the air-tubes, meets with some obstruction, which on inspiration amounts to about half a millimetre of mercury, and on expiration from 2 to 3 millimetres. Thus an oscillation in pressure is produced during the different phases of respiration, which amounts to 2 or 3 millimetres of mercury; that is, 1½ to 2 inches of water. This is called the respiratory oscillation.

If the movements of respiration were left out of account, the intrapleural tension would be equal to that of the atmosphere, minus the elasticity of the lungs; that is to say, it would always give a negative reading on the manometer. It would then be equal to the elasticity of the lungs with the sign changed, that is, − 6 to − 8 millimetres of mercury.

During ordinary respiration the intrapleural tension is also negative throughout; for, if it is negative when the air is stationary, it will be more negative still on inspiration, the lung being more on the stretch; and during quiet expiration, even when from the normal elasticity of the lung the 2½ to 3 millimetres of mercury be deducted which represent the obstruction in the tubes to which the air is subject on expiration, there are still left 4 to 5 millimetres of negative pressure.

During violent expiration, of course, the pressure may rise considerably, even to so much as 70 to 100 millimetres of mercury (3 to 4 inches); but it must be remembered that under normal conditions this pressure does not fall directly upon the pleura, but is immediately supported by the chest walls. Under pathological conditions, on the other hand, when the two layers of the pleura are not in contact, but are separated by air or by fluid, pressure of any kind will make itself felt directly by the contents.

There are two methods of determining the value of the intrapleural tension. 1. In the one the elasticity of the lung is determined, and the result, with the sign changed, is transferred to the pleura; 2, in the other the intrapleural tension is estimated directly by means of a trocar introduced between the layers of the pleura.

In man both these methods of investigation are available after death, but the latter only during life, and this under pathological conditions.

In either case the reading is made upon a mercury- or water-manometer. Water has been more commonly employed, because the oscillations are larger and are more easily read; but the conversion is easily made from the one to the other: thus 1 inch is equal to 25 millimetres, and 1 millimetre of mercury is approximately equal to half an inch of water or 12½ millimetres of water.

Intrapleural tension is often spoken of as "intrapleural pressure," and thus confusion is introduced both in thought and in expression. This confusion will be avoided if it be remembered that the values stated are not actual pressures but readings on the manometer. For instance, if the pressure in the pleura were equal to that of the atmosphere it might be called 1, but as this would be indicated on the manometer by the position of equilibrium which is marked zero, it is usually spoken of as zero; 1, 2, or 3 inches or millimetres would then represent 1, or 2, or 3 above or below the atmospheric pressure, as the case might be.

The elasticity of the lung was estimated by Donders to be from 6 to 8 millimetres of mercury; this, therefore, with the sign changed, would represent the intrapleural tension.

An important series of observations of a similar kind was made by

Perls (2). After a tube connected with a manometer had been fixed into the trachea, first one pleura and then the other was opened and the pressures registered. The observations were made upon the dead body of a man under a variety of different conditions, and the results are very interesting.

Seeing how closely intrapleural tension is connected with the elasticity of the lung this will be the natural place to consider various pathological conditions under which the normal elasticity of the lung is altered.

When one pleural cavity is laid freely open to the air there will then be atmospheric pressure on both sides of the visceral pleura; the elasticity of the lungs will come into play, and the exposed lung will collapse. But this is not all, for the alterations in pressure do not affect the one lung only; the mediastinum being not a fixed partition, but a movable one, the elasticity of the opposite lung also comes into play; with the result that the mediastinum and the organs therein are drawn over to the sound side. Thus it follows that the opening of one pleura not only satisfies the elasticity of the one lung, but goes a long way to satisfy the elasticity of the other. If, for example, the pressures be reduced to figures, and we assume for the sake of illustration that in a healthy man the total elastic contractility of the two lungs together amounts to 50, the opening of one pleura may satisfy this elasticity to the extent of 40, leaving only 10 for the unsatisfied elasticity of the opposite lung.

Thus, in pneumothorax, which is the corresponding pathological condition, if the lungs are healthy and their elasticity at its maximum, the total respiratory capacity will be suddenly reduced by four-fifths. If, however, the lungs be previously diseased or the pleura adherent, the elasticity of the lungs will be either reduced or prevented from coming into play; and thus the change in respiratory capacity consequent on the pneumothorax will not be so extreme. For these two reasons it is evident why the sudden admission of air to the pleura should produce more severe results in a healthy person than in one whose chest has been previously diseased; and a clinical paradox is explained.

Where the pleura is completely adherent the elasticity of the corresponding lung may be almost abolished; but it is frequently retained, though of course when retained it is unable to come into play. Under these conditions the opposite lung often becomes greatly enlarged. This has often been called "complementary emphysema," but in these cases the elasticity of the enlarged lung is not diminished, as in ordinary emphysema, but actually largely increased, so that the elasticity of that one lung may be almost equal to the combined elasticity of two healthy lungs. Thus it is made evident that this condition is not emphysema, but hypertrophy, as there are also the best of clinical reasons for maintaining. It should therefore be called, not complementary emphysema, but complementary hypertrophy.

There is good ground for believing that the contractility of the lung is not simply elastic, but is due in some measure to the muscular fibre with which it is so richly provided. If that be so, we may fairly speak of "pulmonary tone" in the same way as we speak of "vascular tone"; and we may expect it to vary not only with local conditions of nutrition in the lung, but also with defects of nutrition which are general.

Thus, in various local affections, of which pneumonia is the most important, Perls found the elasticity of the lung greatly reduced; as well as in general diseases without any local affection of the lung, as for example in typhoid fever, delirium tremens, erysipelas, phosphorus poisoning, and after severe hæmorrhage.

If, then, pulmonary tone be not simply elastic in origin, but in part neuro-muscular, the loss of it may be met with under two different clinical conditions: first, as the result of general causes—as an evidence, for instance, of general neuro-muscular failure; and, secondly, as a result of local nutritive-disturbance.

As a neuro-paralytic phenomenon it might be placed in association with the like condition in the abdomen (acute tympanites), which in the same way may be due to general or local causes. For example, just as in pneumonia, acute abdominal tympanites may suddenly manifest itself—a phenomenon of fatal significance; so with typhoid fever, or any other specific fever, a similar condition may appear in the lung which is likewise of fatal import.

The loss of pulmonary tone is indicated during life, just as it is after death, by change in the percussion note; the resonance becoming more tympanitic and of that character which is generally known under the name of "skodaic resonance." Without any local disease of the lung, I have on several occasions seen this acute pulmonary tympanites set in; whatever the explanation of its occurrence, there is no doubt as to the existence of the condition.

Where there is local disease in the lung, the other parts of the lungs, as is well known, frequently yield a tympanitic percussion note. There are several conditions under which this is met with: the commonest and easiest to explain is that which occurs with pleural effusion, when the lung floating on the fluid yields this skodaic resonance. The conditions and the percussion note are the same as are presented by the lung removed from the body.

With complementary emphysema, where one part of a lung is diseased and the other parts proportionately distended, similar hyper-resonance is obtained. In this case the hyper-resonance is due to the over-distension of some of the air-vesicles.

But besides these there is another condition which requires a different explanation. Nothing is commoner in pneumonia than to find the parts of the lung above or in front of the affected portion yielding a highly tympanitic note; yet the pneumonic portions of the lung are certainly not collapsed or smaller than they should be, nor are they much larger: thus neither of the explanations just given is applicable; the part of the lung where the hyper-resonance is obtained is not collapsed on the one hand, nor over-distended on the other. This condition, it appears to me, can only be explained on the assumption of loss of lung-tone of neuro-paralytic origin and dependent on nutritive disturbance. This view also obtains support from some of Perls' observations, for among his cases are several instances of pneumonia as well as some of embolism and gangrene; and in all of them the elasticity of the lung was very greatly reduced.

It is possible that the elasticity of the lung diminishes after death, but there are no direct observations to prove this. We may assume at any rate that for some hours after death the elasticity of the lungs is not materially affected.

In estimating the elasticity of the lung and the intrapleural tension the condition of the abdominal muscles and of the diaphragm must not be overlooked. We have to reckon on the one hand, during life, with their respiratory action, and on the other, after death, with rigor mortis; but it is not necessary here to do more than refer to these complicating factors.

THE PLEURAL CAVITY UNDER PATHOLOGICAL CONDITIONS

Under pathological conditions the two layers of the pleura may be separated either by air or by fluid, and each of these presents its own peculiarities and difficulties: thus, fluid has weight, but is practically incompressible; air is compressible, but its weight may be disregarded. With fluid, therefore, the height of the column above the point of the trocar will affect the manometer readings, while with air the position of the trocar is immaterial. As in many respects the problem is simpler in the case of air than of fluid, it will be well to begin with pneumothorax.

Intrapleural tension in pneumothorax.—Air may gain access into the pleura either from without through the chest walls, as by a wound, or internally from the lung; and in both cases we have to consider, first, the condition in which the air enters more freely on inspiration than it finds issue on expiration, and, secondly, the condition in which there is no abnormal obstruction either on inspiration or on expiration.

A. Where the air finds entrance through the chest walls.

(i.) *By a punctured wound.*—In this case, where the wound is a small one and merely a puncture, though the lung be injured the air does not, as a rule, find access to the pleura, but crosses the pleura and reaches the subcutaneous tissue. The reason of this is very difficult to find, but of the fact there is no doubt; it need not, however, be considered here.

(ii.) *Where the opening is a small one*, so that the air finds easier entrance than it finds exit. This condition will be the same as that in which the air gains access to the pleura through the lung, and will be better considered later.

(iii.) *Where there is a large opening through the chest walls*, at least as large as the cross-section of the trachea. The air then enters and leaves the pleura without obstruction, that is to say, the pressure on both sides of the visceral pleura is the same—namely, that of the atmosphere—during all phases of respiration. Under these circumstances the elasticity of the lungs comes simply into play, so that the lungs collapse.

It is no doubt true, as Donders said, that in course of time under these circumstances the lungs will become completely collapsed by virtue of their own elasticity; yet we have daily experience that this does not usually occur, and when we consider the matter the reason is clear. It is found in the fact which has already been stated; namely, that the air in the tubes is not subject simply to the atmospheric pressure during the phases of respiration; on inspiration it is under a pressure somewhat less than the atmosphere (by half a millimetre of mercury), and on expiration under a pressure above that of the atmosphere (to the extent of 1½ to 2 millimetres of mercury).

During expiration, therefore, the lungs will always be subject to the distending force of 1½ to 2 millimetres of mercury. There are no observations to show how far the lung will be expanded under such a pressure, but it cannot well be less than a half, and is probably more; at any rate we have daily demonstration of the fact that the lungs do not collapse completely as the result of opening the side: on the contrary, on opening the side for empyema it is a common experience to find the lungs which have been completely collapsed by the effusion expand again as soon as the pus is evacuated, so as to reach close to the chest walls immediately after the operation. This may at first be the result of the violent respiratory efforts or of the coughing which very frequently follows the operation; but this is not the only explanation, for it occurs when there is no violent expiration or coughing, or persists when they have passed off.

Two cases which I have recently recorded are of interest in this respect, because the lung had been compressed by fluid for a long time—eighteen months and five months respectively, one being a case of serous effusion and the other of pyopneumothorax; in both, immediately after the operation, the lungs were close to the chest walls, and within a week had come into close contact with it everywhere except just round the incision (3).

B. Where the air gains access to the pleura from the lung.

(i.) Theoretically it is possible that the opening through the lung should be large enough for the air to pass freely in and out during inspiration and expiration without obstruction; yet this is a condition which can hardly ever arise, and almost all the cases of pneumothorax therefore come into the second category.

(ii.) That in which the opening through the lung is of such a kind that though the air gains free entrance into the pleura during inspiration it cannot find free issue from it during expiration. The result of this is that during expiration the pressure rises and compresses the lung, which gradually becomes more and more collapsed. Although it is true that the mediastinum may be displaced to the maximum and the lungs be completely collapsed in cases where there is no expiratory compression, still in the great majority of cases this rise of pressure during expiration plays a very important part in the production of both these phenomena.

The division of pneumothorax into open, closed, and valvular, interesting as it is in some respects, is of no practical importance from the present point of view—that of intrapleural tension; for in a case of recent pneumothorax as soon as the lungs are completely collapsed the hole becomes closed, whether it be permanently sealed or not. During the early stages pneumothorax is always more or less valvular; in other words, the air finds easier access during inspiration than it finds issue during expiration.

The intrapleural pressures during inspiration and expiration require, in the case of pneumothorax, to be considered separately.

1. *The inspiratory pressure.*—When the lung has ruptured, air finds access to the pleura during inspiration so long as the pressure in the pleura is below that of the pressure in the air-tubes; that is, below the atmospheric pressure:—although this has to be reduced, as already stated, by half a millimetre of mercury, being the value of the obstruction which the air meets with on its way into the lungs. The inspiratory pressure, therefore, can never rise in pneumothorax above that of the atmosphere except under one condition, namely, that in which there has been much dyspnœa; for as then the inspiratory efforts are considerable the air will consequently continue to enter the pleura as long as the pressure at the end of each inspiration is below that of the atmosphere and until it equals that of the atmosphere, after which no more air can enter. It follows, therefore, that if the patient survive and the dyspnœa pass off, the inspiratory pressure might be above that of the atmosphere to the extent of the difference between the pressure on deep inspiration and the pressure on ordinary inspiration. This is not very much, and in all probability the excess of air, which represents the difference of pressure, is rapidly absorbed.

In ordinary simple pneumothorax the inspiratory pressure is therefore not, as a rule, above that of the atmosphere. If it be, some other factor is required to account for it, and this almost without exception proves to be the presence of fluid; we may therefore conclude that whenever the inspiratory pressure is much raised we shall probably find that fluid is present as well as air.

2. *The expiratory pressure.*—The expiratory pressure in pneumothorax is always positive. It is true that the mediastinum may be displaced to its maximum in a case where the pressure in the pleura is zero. Still the raised expiratory pressure tends to make the displacement extreme or to produce it more rapidly; while, as already stated, it is the expiratory pressure which probably chiefly accounts for the complete collapse of the lung.

3. *The respiratory oscillation.*—As this is the difference between the pressure on inspiration and the pressure on expiration it will vary according to the amount of dyspnœa or the violence of the respirations at any given time.

It might be thought, considering the violence of respiration in many of these cases, that the respiratory oscillations would always be considerable. As a matter of fact this is not found to be so, and a little consideration will show why this is the case; on the affected side the chest is in a condition of maximum inspiratory expansion and cannot alter from this on expiration; while on the opposite side the lung is prevented from expanding fully, by the amount of the reduction of its volume on the displacement of the mediastinum and the organs connected with it; thus its elasticity also is reduced, being, as already stated, partly satisfied. It is evident, therefore, that the total respiratory excursion of the chest will be very considerably diminished and the respiratory oscillation therefore small.

In a recently published paper (4) I have recorded a series of observations upon the pressures in pneumothorax in eleven cases, some of which were tapped several times; so that there are records of twenty different paracenteses.

The *inspiratory pressure* varied from zero to + 9, the several pressures being 0, ½, 1, 1½, 2½, 4, 4½, 5, 6¼, 6½, 6¾, 7, 8¼, 8½, 9.

In two cases the inspiratory pressure was that of the atmosphere; that is, the reading of the manometer stood at zero. In both of these cases fluid was present as well as air. From this it is evident that as soon as the fluid formed the air must have been absorbed, since the opening into the lung in both cases was closed.

In another case the inspiratory pressure, after having been in the two first paracenteses positive, fell in the last two to zero; and the change in pressure was due to an opening of considerable size having formed into the lung.

In all the other cases the inspiratory pressure was positive, and fluid (sometimes pus, sometimes serum) was present as well as air; thus the statement already made is confirmed, namely, that when the inspiratory pressure is much above that of the atmosphere the conclusion may be drawn that fluid is present as well as air.

It is no matter of wonder that the inspiratory pressure rises when fluid forms; but it is surprising that the pressure is not much higher than we find it. The highest pressure that I observed was nine inches of water, but pressures as high and even higher have been met with in serous effusions. It follows, therefore, that when fluid forms in pneumothorax a large amount of the air present must be absorbed as the fluid forms.

We know, both as the result of experiments on animals and of operations upon man, as well as from observations of pneumothorax in man, that air may be very rapidly absorbed from the pleura.

Even when fluid is present the pressure may not be above that of the atmosphere, as we have already seen; and I think we may possibly even go so far as to say that if the intrapleural pressure remains unusually high in pneumothorax it may be taken as an indication that there is extensive disease both of the lung and the pleura; so that the absorption of air which would ordinarily occur is prevented from taking place.

The *expiratory pressure* also varied considerably from zero up to 13¼, the actual figures being 0, 0, 1, 1½, 2¼, 2½, 4½, 5, 7, 8, 8½, 9, and 13¼. The highest expiratory pressures are, as already stated, due to dyspnœa; that is, to violent expiratory efforts.

The *respiratory oscillations* in the same way showed great variations, and fluctuated from zero up to 8. The largest were 8, 6⅝, 6½, 6, and 4. In all these cases there was dyspnœa, and the large respiratory oscillation was the result of the high expiratory pressure.

The lower respiratory oscillations were 0, ½, ¼, 1, 1½, 1¾, and 3¼.

Even where the inspiratory and expiratory pressures are high, the respiratory oscillations may be small or absent; thus in one instance where the inspiratory pressure was + 9, the expiratory pressure was the same, and the respiratory oscillation therefore 0. *Per contra* even where the inspiratory pressure is low, the respiratory oscillation may be considerable if there be much dyspnœa; for example, in a case in which the inspiratory pressure was 0, the expiratory pressure was + 8, and the respiratory oscillation therefore 8.

Where there is no dyspnœa the respiratory oscillations are apt to be small, and may be completely absent.

These observations show that, in pneumothorax, whatever general statements may be made, they have to be applied with caution in individual cases, for it is impossible in any given case to forecast what the actual pressures will prove to be; and, finally, that although the results obtained will have to be explained according to the peculiar circumstances of each case, yet if this be done carefully, much information may be obtained concerning the actual condition of the lung and pleura.

Intrapleural tension in serous effusion.—In health the pleural cavity contains no fluid, and we often speak of it as dry; yet this description is somewhat inaccurate, for there is in fact a constant circulation of fluid into the pleura and out of it, the fluid being effused by the blood-vessels and carried away by the lymphatics. The mechanism by which this is performed has been described as "the lymphatic pump." It consists of the lymphatic vessels with their stomata and valves, and is worked by the respiratory movements. The course of the circulation in the lung is from the pleural surface towards the root of the lung, as has been determined by experiment; and there is a similar circulation from the pleural surface through the diaphragm and through the chest walls. It is partly through the action of the lymphatic pump that the negative pressure is maintained in the pleural cavity and the lungs kept fully expanded.

There are two ways, therefore, in which fluid may accumulate in the pleura: either it may be poured out into the pleura in larger quantities than the pump can remove, that is, its amount may be abnormal, or the amount of fluid not being above the normal, the pump may be defective.

In the case of pleural inflammation both these processes come into play; the amount of transudation is considerable, while the stomata and smaller lymphatics are often plugged by deposits of fibrin. Thus in inflammatory cases the fluid may accumulate with very great rapidity and soon reach a large amount.

In the case of dropsy of the pleura consequent, let us suppose, on heart disease, the explanation is probably also in great part mechanical. Exudation under these conditions takes place from the blood-vessels into the lymphatics of the lung, which become water-logged or choked; thus it is unable to carry off the fluid from the pleural cavity, which consequently accumulates in it. With dropsy, however, the accumulation of fluid is much slower and the amount as a rule much less.

When fluid collects in the pleura it falls by its weight to the lowest part; and although the tension in the whole pleural cavity is diminished in proportion to the amount of fluid present, still the effect upon the different parts of the lung is different: thus the lowest parts suffer most and become collapsed, while the upper parts of the lung remain distended; yet the tension in the upper part of the pleural cavity is also lower than it otherwise would be, as is shown by Calvert's observations. The diminished tone in the lung or tension in the pleura explains the hyper-resonant note which is obtained in those parts of the lungs which are floating upon the fluid.

In determining the intrapleural pressure in cases of fluid effusion something will depend upon the seat of puncture. This Calvert has also demonstrated; for if the mouth of the trocar be 1, 2, or 3 inches respectively below the level of the fluid, there will be the pressure of a column of fluid of this height to allow for. If, for example, the intrapleural tension be equivalent to – 3 inches of water, and the amount of fluid exuded into the pleura be sufficient to reduce this 3 inches negative pressure to 2 inches negative pressure, it follows that if the mouth of the trocar be 2 inches below the level of the fluid, a positive pressure of 2 inches will have to be added to the negative pressure in the rest of the pleura, which will reduce the pressure-reading to zero; or, if the height of the fluid be 3 inches instead of 2 inches, it would convert the pressure at the point of puncture to a positive pressure of 1 inch. It is very difficult to make due allowance for these variable conditions, so that the pressure records in pleural effusions have not anything like the same value as those in pneumothorax.

It might be supposed at first that with large effusions the pressure would be high, with medium-sized effusions moderate, and with small effusions low; but actual observation shows that this is by no means the case, for whatever be the bulk of the effusions the pressures may be high, moderate, zero, or even below zero. Thus, among my own observations, where the effusion was large and a considerable quantity of fluid was drawn off, the pressures were – 1, 2½, 4, 6, 8, 11½, and 18; where the effusion was moderate – 1, 0, 4, 5, 8½; and where it was small, 0, ½, 1¼, 3, 5, 11.

The pressures, therefore, vary in a curiously irregular way, and it is difficult to see what the explanation can be. It is natural to attempt to refer these variations to the different stages of the inflammation. Thus in the early ingravescent stage, when the effusion is rapidly forming, the pressures might be high, and low in the later stages when the fluid is being absorbed. There is some evidence in favour of this view, but the matter is by no means as simple as it would seem.

Respiratory oscillations.—For the reasons given when speaking of pneumothorax the respiratory oscillation with serous effusion is likely to be small; as a matter of fact it is so, and not infrequently it is entirely absent.

Now, as the action of the lymphatic pump depends upon the respiratory movements, and as these are indicated by the respiratory oscillations, it is evident that in these cases the mechanism for the removal of the fluid is at a standstill.

It is interesting to observe in some cases, though the respiratory oscillation is absent when the puncture is first made, that after fluid has been withdrawn the respiratory oscillation begins to return, and at the end of the operation may be fairly considerable. This is important, as it explains what is often observed at the bedside, namely, that the removal of a small quantity of an effusion may lead to the rapid spontaneous disappearance of the rest. What it really means is, that the lymphatic pump has been set to work again.

The intrapleural pressure in serous effusions is the resultant of three forces:—1. The respiratory movements. The effect of these has been already sufficiently considered. 2. The force of inflammatory exudation. We do not know much of the pressures under which the exudation of inflammatory fluid takes place in the pleura; but if we may compare it with the knee-joint, which is more accessible to observation, we may be quite sure that it occurs under very considerable pressure when we remember how tense the synovial sac becomes during the early stages of inflammatory effusion. 3. The action of the lymphatic pump is opposed to the first. We may presume that it is practically equivalent to the elasticity of the lung, and therefore equal to 6 or 8 millimetres of mercury, when the lung is fully distended; but it is a rapidly diminishing force as the lung becomes compressed, the stomata closed, and the lymphatics collapsed; and when the chest is full of fluid it vanishes, for, as the respiratory oscillations show, the lymphatic pump comes to a stop.

In the early stage of acute inflammation we may conclude that the pressure may be very high when the effusion is a large one, or when the effusion, if a small one, is encapsulated, that is, localised and not general.

When the acute stage of the inflammation has passed and exudation ceases, if the fluid begins to be slowly removed the pressure will fall; and it is obvious, since the fluid is ultimately removed completely, and the lungs come out into contact with the chest walls again, that in course of time the pressure will even become negative. I do not see any way in which this can be brought about except through the intervention of the lymphatic pump.

Intrapleural tension in empyema.—This is a much simpler problem than in the case of serous effusions. The pressures here are in accord with what we know of suppuration elsewhere; for the formation of pus goes on under considerable pressure. It is only in the very chronic so-called "cold" abscesses that the tension is low; but even then the pressure is probably above that of the atmosphere.

Thus among my own observations the pressure was considerably raised in all cases, the lowest being + 3. The highest was + 16, and this was found with a very large effusion; but, as I have said, small effusions may have a very high pressure if they be loculated or encapsulated. An interesting example of this was observed among the cases of serous effusion; for in one in which the pleura had been tapped twice, and the pressure found on each occasion to be not raised, on the third paracentesis the pressure was + 3; the effusion, however, was no longer serous, but had become purulent: in other words, the general serous effusion had been followed by a small localised empyema; this was incised and then recovery became complete.

The respiratory oscillation in empyema is always small and frequently entirely absent.

From what has been said it is evident that the problem of intrapleural tension, especially under pathological conditions, is a very complicated and difficult one, and requires much further investigation.

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S. W.

PLEURISY

THE name Pleurisy (*ἡ πλευρῖτις νόσος*, morbus lateralis, side-sore of early English) formerly denoted that acute disease which is characterised by fever and severe pain in the side; and the meaning of the word was wholly clinical. After the time of Morgagni, when the influence of morbid anatomy became predominant, the name acquired that anatomical signification which it has since retained; and for the last century or more, pleurisy has been defined to mean inflammation of the pleural membrane.

I. Ætiology.—1. *Age and Sex.*—Pleurisy occurs at all ages: I have evacuated pleural empyema in infants aged one month, three months, and five months; and I have drawn off three pints of serum from the chest of a woman eighty-seven years old. The annexed table, drawn up from the records of St. Bartholomew's Hospital for ten years (1884-1893), shows the number of patients treated for pleurisy, and in whom pleurisy was the main and foremost disease: it does not include the cases in which pleurisy was secondary to some other disease no less serious. The figures show: that pleurisy is much more frequent in males than in females; that pleurisy with effusion of coagulable lymph or of serum (dilute liquor sanguinis) is most common in patients between twenty and forty years old; and that pleural empyema is most common in patients less than ten years old.

Effusion.	Males.	Females.	Deaths.	5 yrs. and under.	10 y.	15 y.	20 y.	30 y.	40 y.	50 y.	60 y.	Over 60.	Totals.
Not purulent	465	186	48	25	59	50	54	179	149	85	35	15	651
Purulent	155	61	48	53	32	15	22	48	17	23	6	0	216
Totals	620	247	96	78	91	65	76	227	166	108	41	15	867

2. *Specific Poisons.*—Pleurisy is due to irritation of the pleural membrane by certain morbific microbes or poisons. It is difficult not to believe that this proposition is universally true; and true, even in the case of pleurisy following upon an injury to the side, or upon exposure of the chest to cold. (i.) A heavy blow upon the chest, not leading to more than bruising of the parts, and not bringing about any solution of continuity, will sometimes be followed by constant pain, and at length by serous effusion into the pleural cavity: in such a case it is reasonable to suppose that the injury affords an opportunity for infection. (ii.) Again, pleurisy, like pneumonia, will sometimes follow so speedily upon great exposure of the whole body, or of the chest in particular, to cold, that it is carrying scepticism to excess to doubt that the exposure has something to do with causing the subsequent disease: in this case, also, the cold may be supposed to bring about such an altered nutrition of the parts as favours invasion by specific microbes.

But in by far the greater number of cases pleurisy is spontaneous, and arises apart from the operation of any obvious antecedent cause. Microbiology has thrown great light upon this spontaneous or idiopathic pleurisy.[1] The microbes which will account for most pleurisies are three,—tubercle bacillus, streptococcus, and pneumococcus.

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[1] The following details concerning bacteriology are taken from an article by Dr. Netter in the 4th vol. of the *Traité de médecine*, edited by Charcot, Bouchard, and Brissaud.

(i.) Tubercle bacillus.—(α) Even before the discovery of the bacillus of Koch, it was suspected that many cases of pleurisy with serous effusion were due to tuberculosis of the pleura. But now it cannot be doubted that tubercle is the commonest cause of pleurisy with serous effusion, an opinion supported by the following facts:—Many of the patients have inherited a tendency to tuberculous disease. Some of them have suffered from manifest tuberculous disease before the pleurisy began. Many of those who die are found by examination post-mortem to be tuberculous. Many of those who recover from the effusion suffer afterwards from tuberculous disease, and especially from pulmonary consumption. On the other hand, it is admitted that, even in cases which are undoubtedly tuberculous, bacilli are seldom found in the effusion, and cultivation of the fluid gives no result. Inoculation of the pleural serum into the peritoneal cavity of guinea-pigs is more successful; many of the animals are infected thereby. (β) Purulent effusion is less often dependent upon tubercle. Empyema in a tuberculous subject is sometimes due, not to the tubercle, but to streptococci or pneumococci: the distinction depends upon microbiological examination.

(ii.) Pyogenetic streptococcus is the microbe most commonly found in the pleural empyema of the adult. The morbific germ reaches the pleura:—(α) Through the lung, in pneumonia, dilated bronchi, gangrene, pyæmic abscess, tubercle, cancer; (β) through mediastinal organs, in pericarditis, disease of œsophagus, abscess spreading from neck or throat; (γ) through walls of chest, in penetrating wounds, abscesses, lymphangitis, disease of breast, and especially cancer; (δ) from caries of vertebræ, which is sometimes quite latent; (ε) through peritoneum, in peritonitis, subphrenic abscess, suppuration of liver or spleen; (ζ) through the blood, in general diseases, scarlet fever, diphtheria, erysipelas. (η) Adjoining local disease sometimes seems to act as a mere irritant of the pleura, and so to render it susceptible to purulent infection by the blood: aneurysm of the aorta is an instance of this kind, the aneurysm itself being possibly quite latent.

(iii.) Pneumococcus is the microbe most commonly found in the empyema of childhood. In most of the cases it cannot be proved that pneumonia preceded or accompanied the empyema, and this is especially true with respect to children. In other words, primary pneumococcous pleurisy is a common disease. When secondary to pneumonia there is usually an interval of some days' duration between the defervescence of the pneumonia and the occurrence of the symptoms and physical signs of pleural effusion; but sometimes there is no interval, the empyema begins before the pneumonia has ended; on the other hand, the interval is sometimes much longer, several weeks or months. The pleuritic effusion which is subsequent to pneumonia is not always purulent, but is sometimes serous: in this serous effusion pneumococci are found. Moreover, serous effusion, which is not secondary to pneumonia, is due in a few cases to pneumococci.

(iv.) There are some other causes of pleurisy, but the specific manner in which they operate has not been discovered: such are superficial hæmorrhagic infarctus of the lung, nephritis, rheumatism, and gonorrhœal rheumatism.

II. **Symptoms,** that is to say, *signa assidentia*, are the signs which are not pathognomonic or characteristic of the disease. Yet inasmuch as these are the signs which the patient recognises, and which are, therefore, the signs first recognised, they may be aptly discussed in the first place.

1. *Onset of the disease.*—(i.) Latent.—The occurrence of one or more of these symptoms marks the onset of the disease in most cases. But in other cases the onset is not perceived: the disease at first is latent; and it is most likely to be latent when it is secondary to other serious disease, the symptoms indicative of the onset of pleurisy being masked by pre-existing symptoms dependent upon the primary disease.

(ii.) Manifest.—When the onset of the disease is not latent, the indicative or invasion symptoms either (*a*) occur suddenly and decisively, clearly marking the time at which the state of health passes into the state of sickness; or (β) they occur gradually, so that it is not easy to say precisely when the disease began. Whether they occur suddenly or gradually, these symptoms, denoting the onset of the disease, are no other than more or fewer of the symptoms which attend the confirmed disease, and which will be described in the next place. The commonest invasion symptoms are fever (with shivering or not), pain in the side, vomiting, cough, quickness and shortness of breathing; in children sometimes convulsions.

2. *Fever.*—Fever is not a constant symptom; being slight and temporary in pleurisy, with small innocuous exudation; being present in most, and yet not in all cases of larger effusion; being absent sometimes even in empyema.

(i.) In acute pleurisy the temperature seldom rises above 103°. In pleurisy, as compared with pneumonia, the fever is not so high, shivering at the onset is less common, and the duration of the fever is indefinite.

(ii.) In chronic pleurisy no distinction can be drawn between serous and purulent effusion by means of the characters of the fever. (*a*) What is called serous effusion is not serous in the strict meaning of that word, but is really a dilute lymph or liquor sanguinis[1] not free from leucocytes. In pleurisy with serous effusion the temperature is often (but not always) persistently raised; and the fever not less or less constant than that of a purulent effusion. When fever is present it lasts until the effusion is wholly absorbed; indeed, in cases of febrile serous effusion defervescence is the best evidence that the effusion has been absorbed, for physical examination is often of no avail in determining this point. The type of fever tends to be quotidian remittent. (β) In pleural empyema, when the pus is pent up or offensive, the temperature will be raised almost for certain. Evacuation of the pus will be followed by defervescence, temporary or permanent. In fistulous empyema the temperature is usually almost or quite natural; and a rise of temperature means that pus is pent up somewhere. The type of temperature tends to be that of septic fever, namely, quotidian remittent with evening exacerbation. Colliquative symptoms (heavy sweats, and especially diarrhœa) sometimes attend the fever. Lastly, in some cases of small empyema, even when undrained, the temperature remains normal.

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[1] A fact first recognised by B. G. Babington in 1830: see *Med.-Chir. Trans.* vol. xvi. p. 303.

Local temperature.—That the affected side is sometimes hotter than the other was known to ancient Greek physicians, who employed an ingenious means of discovering the fact (see Hippocrates, *De Morbis*, iii. chapter 16).

3. *Pain.*—Severe pain in the side was the main and constant sign of the disease called pleurisy in the ancient sense of the word; but the pain of pleurisy, in the modern sense, may be severe, or may be not severe, or there may be no pain at all. The pain is usually felt in the side of the chest; sometimes about the nipple, or above the clavicle, or in the hypochondrium; sometimes about the navel, or even in the iliac fossa and lower belly, on the same side as the disease. The skin over the affected side is often very tender. Marked spinal tenderness, in some part of the vertebral groove in the dorsal region, is common.

The nature of the pain is a matter for debate; probably there are different causes of the pain. Intercostal or diaphragmatic cramp has been suggested as an explanation of the stitch in the side. The pain felt at a distance (namely in the abdomen) is probably conducted along an intercostal nerve.

4. *Dyspnœa.*—Dyspnœa, manifested by frequent or laboured breathing, is common. Patients kept in bed are apt to become accustomed to the want of breath, and so their dyspnœa may diminish or even disappear; although the quantity of pleural effusion (if present) remain unchanged. Dyspnœa is in some cases greatly due to associated disease, for instance, to chronic pneumonia on the same side as the pleurisy. The dyspnœa of pleurisy without liquid effusion is chiefly shortness of breath; that is to say, inability to breathe freely and deeply because of the pain caused thereby.

5. *Cough and Expectoration.*—Cough is usually present; but in rare cases there may be no cough, even in pleurisy going on to effusion.

Concerning the sputa.—The terms dry and humoral pleurisy, in the old sense of these words, relate to the absence or presence of expectoration. For sometimes there is no expectoration, and therefore no exspuition. But commonly there is expectoration, although the humours coughed up are not always spat out. (i.) The sputa sometimes consist of mucus nearly pure, judging from their colour and transparency: when the mucus is thin and watery, like gum water, it is called pituitous. (ii.) More often the sputa are muco-purulent. (iii.) More or less blood in the sputum is not uncommon at the onset of the disease. (iv.) When an empyema has burst into a bronchus the sputa are almost pure pus. (v.) It is a very uncommon event for a serous effusion to burst through the lung, and so to be expectorated. Yet this seems to have happened in a case narrated by Dr. Vincent Harris in *St. Barthol. Hosp. Reports*, vol. xxiii. p. 34. Less uncommon is the muco-serous (or albuminous) expectoration, which sometimes occurs during or soon after paracentesis thoracis, and which will be described in connection with that operation (p. 376). (vi.) Fœtid expectoration depends upon one of two conditions: either a fœtid empyema has burst through the lung—by far the more common case; or a fœtid empyema, which has certainly not burst into the lung, communicates an offensive smell to the secretions of the air-passages in the neighbourhood, just as abscesses near to the alimentary canal often acquire, for this reason, a disgusting smell.

6. *Vomiting and Diarrhœa.*—Vomiting is common at the onset, especially in children.

Diarrhœa is common in pleurisy with effusion, serous or purulent. Diarrhœa sometimes occurs from the very onset of purulent pleurisy: should vomiting, pain referred to the belly, and tenderness of the belly concur with diarrhœa, peritonitis will be closely simulated at first sight. This diarrhœa tends to be very obstinate, and in many cases cannot be stopped until the empyema is cured. Diarrhœa and marasmus may be the main symptoms of a small empyema. Should the patient die, post-mortem nothing amiss with the intestines will be discovered by the naked eye: it is a septic diarrhœa.

7. *Septic infection of whole body.*—(i.) Symptoms which are called typhoid or putrid, and which are indicative of septic infection of the whole body, are apt to accompany fœtid empyema. The tongue is dry and brown, the secretions become offensive to smell, the eyes are yellowish, the face is dusky, the pulse soft and weak, consciousness blunted, and muscular debility, or prostration, great. (ii.) Like symptoms sometimes occur from the very onset of empyema which is not fœtid. In a state of good health sudden shivering occurs, headache, cough, in some cases much pain in the side; in others no pain at all. The fever is high, the temperature often reaching 104° or more; respiration frequent; sputa not rusty. Consciousness becomes affected; in some cases so much that even as early as on the second day the patient is deeply comatose; but the degree of coma is apt to vary, so that the patient, after deep unconsciousness, may become fairly sensible. More or less delirium occurs in some cases, but in others none at all. Morbilliform mottling of the skin (not much like the rash of typhus); temporary redness, swelling, and tenderness of one or several joints; enlargement of the spleen and diarrhœa may occur in some patients. The urine may be albuminous or not. The physical signs of effusion are sometimes late to appear, and are apt to be mistaken for those of pneumonia. The patient will die within ten or twelve days; and whether paracentêsis be employed or not seems to make small difference. The pus

has been found to contain pneumococci (21); but pneumococcous pleurisy is seldom attended by these grave signs of universal poisoning.

8. *Latent pleurisy.*—Pleurisy is sometimes latent, in the sense that the symptoms of the disease are slight, nay almost absent; and this even in the case of large effusion. But it is only in the neglect of physical examination that pleurisy, unless its extent be very small indeed, can ever be really latent.

III. **Signs.**—Signs which are pathognomonic, signs by which pleurisy can, with certainty, be distinguished from other diseases, are of two kinds; namely, physical signs and the result of puncture. These signs do more than this; they enable us to distinguish two kinds of pleurisy, which it is important should be distinguished; namely, pleurisy with exudation of coagulable lymph only, and pleurisy with liquid effusion. Moreover, puncture enables us to distinguish the different kinds of liquid effused.

A. *Pleurisy with no liquid effusion.*—1. This condition often exists unattended by physical signs of disease, or at most attended by signs which are not distinctive; such as some degree of retraction of the chest, some loss of clear tone on percussion, some weakness of breathing sound.

2. The only sign which is quite distinctive is friction sound. But it is very far from being a constant attendant upon pleurisy, even when the effusion is nothing more than coagulable lymph. Indeed it might be said, and probably with truth, that even under these conditions friction sound is more frequently absent than present. Friction sound is to be recognised by its peculiar friction quality, giving the notion either of rubbing to any degree between lightest grazing and harshest scraping, or of creaking like that of leather. Friction is usually a very local sound, heard over a small part of one side only; and that part is mostly where the rib movements are freest, namely, the lower part of the chest, below the nipple or armpit, or about the angle of the shoulder-blade.

B. *Pleurisy with liquid effusion.*—1. Before the effusion becomes abundant enough to gravitate, a friction sound is sometimes (but seldom) heard. Still more uncommon are signs which attend the onset of pleurisy with effusion in rare cases, and which closely resemble those of bronchitis. The distinction between the two diseases is to be found in the fact that bronchitis very seldom affects one side only, and that pleurisy with effusion very seldom affects both sides. The signs referred to are these:—The affected side moves less freely than the other; the percussion note is raised in pitch and muffled over the greater part or the whole of the side; the sense of resistance to percussion is increased; the breathing sound is weak and attended by widely-spread rale, which is quite indistinguishable from the rale of bronchitis. This rale has been called friction-rale, thereby to indicate the belief that the sound is produced in the pleural sac. But it seems more probable that the rale really is a bronchial and mucous rale produced in the air-tubes, and that the catarrhal or bronchitic state of the lung is due to its relaxation or deficient expansion consequent upon the pleural effusion, small though it be.

2. Much more frequently, however, the earliest signs of pleurisy with effusion are those which indicate that the effusion is already abundant enough to have sunk to the lowest place. What constitutes the lowest place depends upon the attitude assumed by the patient while effusion is going on. At first, when the quantity is small, the lung is simply relaxed by virtue of its own elasticity, and swims upon the effusion; but as the liquid accumulates, it compresses the lung and renders it more or less empty of air.

(i.) The great sign of liquid effusion is a coextensive dulness to percussion. This dulness is not wholly due to the effusion, but is partly dependent upon associated collapse of lung; that is to say, a layer of liquid an inch or more thick would transmit percussion resonance of the lung were the lung resonant. Dulness begins at the lowest part of the chest behind, the note being natural elsewhere. When the effusion has risen higher than the angle of the scapula, the lung will have relaxed to such an extent as to give a clear tracheal[1] note above the nipple of the same side in front—a sign not always present even in cases watched day by day from the onset. Whether, by further increase in the quantity of the fluid, the whole back become dull before the front is so at all, or whether the upper level of the fluid be comparatively horizontal, depends upon the attitude assumed by the patient while the effusion is going on. Hence, when the effusion is small the dulness may be wholly posterior, and sharply defined in front by the posterior axillary line, the lateral region remaining resonant. On the other hand, the upper limit of a dulness which occupies the lower rather than the hinder part of the chest often rises higher in the axillary region than in the back. Even when absolute dulness is confined to the base, there is usually some impairment of resonance all over the back on that side. The dulness over the effusion may be far from absolute. The anterior clear resonance, when present, is sometimes of cracked-pot quality. The effusion, even when partial, does not shift its position easily or at all with changes in the position of the body.

(ii.) In proportion to the amount of effusion the side is enlarged, diaphragm depressed, and mediastinum displaced. (*a*) The side, compared with the other, will possess these characters: shape, on horizontal section, rounder; antero-posterior diameter longer; length from above downwards diminished; shoulder raised; spine curved towards the unaffected side. The antero-posterior enlargement becomes very obvious when the physician stands behind the patient so as to look obliquely over the shoulders and the front of the chest. Circumferential measurements of the two sides are often made for the sake of comparison, but be it remembered that, by the passage of the elliptical form into the circular, considerable increase in the sectional area of the chest may occur, whilst the length of the periphery remains the same. Moreover, the displacement of the mediastinum thrusts the heart into the unaffected side. Add this consideration, too, that the walls of the healthy side must follow the antero-posterior projection of the diseased side; and then it will be plain why, as a matter of fact, the perimeter of the affected side often measures very little more, nay, sometimes even less, than that of the side which is not diseased. The cyrtometer, by indicating shape as well as circumference, affords us the true means of recording the amount of unilateral enlargement. (β) Displacement of the mediastinum is indicated by displacement of the heart. Effusion into the right pleura may displace the heart so as to cause its impulse to be felt in the left axillary line, and in any interspace from the second to the sixth. Effusion into the left pleura may displace the heart so as to cause its impulse to be felt anywhere between its natural position and the right nipple line, and in any interspace from the fourth to the seventh, or in the epigastrium. When the impulse is felt to the right of the natural position, it is often some part of the heart, other than the apex, which strikes against the chest; and this part is usually the right conus arteriosus. When the heart is displaced to the right, there is, in most cases, no considerable change in the relative position of base and apex; that is to say, the heart does not swing to the right upon its base as a fixed point. Yet such a change in the attitude of the heart does sometimes occur, and the very ventricular apex may beat in the right nipple line. The displacement of the heart is often more or less than might be expected; for instance, it may remain unmoved by an effusion of not less than a quart of serum into one pleura. Percussion of the sternal region above the heart sometimes affords evidence of displacement of the mediastinum: the upper part of the sternum naturally yields a clear resonance; under the pressure of a copious liquid effusion into either pleura, the mediastinum bulges so much towards the unaffected side as to afford absolute dulness to percussion in the sternal region, and even somewhat beyond it. (γ) Displacement of the diaphragm downwards is determined by ascertaining the position of the liver, spleen, and stomach. When the quantity of fluid in the left pleura is very great, the left half of the diaphragm may possibly be depressed to such a degree that not only can the lower margin of the spleen be felt, but even its upper margin, in fact its whole outline. At the same time, the thrusting of the mediastinum and heart into the right side of the thorax may depress the right wing also of the diaphragm to an almost equal degree; a point ascertained by examination of the liver.

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[1] What the Germans call "tympanitisch." For the exact meaning of the technical terms used in these pages with reference to percussion and auscultation, I must refer the reader to my book on those subjects.

(iii.) Vocal thrill is diminished where dulness to percussion exists, and is wholly abolished in great distension of the side.

(iv.) The respiratory sound is at first weakly vesicular, and sometimes remains so throughout the disease. But often the breathing soon becomes bronchial, sometimes even before the dulness becomes absolute. With progressive increase of effusion the bronchial breathing tends to become less and less loud, until, at last, it is wholly suppressed. But sometimes, although the quantity of fluid be very great, loud bronchial breathing is heard all over the affected side: the fact being that the loudness depends, not inversely upon the quantity of liquor effused, but directly upon the openness of the air-tubes; for liquid is a good conductor of sound.

(v.) Vocal resonance is weak or bronchial in much the same manner as the breathing sound. When the effusion is partial, with clear resonance in front, the bronchophony is sometimes ægophonic about the angle of the scapula. Ægophony is a sign of little or no value. In the first place, well-marked ægophony is seldom heard; next, it is sometimes heard over simple consolidation of the lung, such as is left by absorption of pleural effusion; and, lastly, ægophony certainly does not always attend thin layers of liquid in the pleural sac.

(vi.) By percussing the chest in front with two coins, and auscultating behind as for the bell sound, a pleural effusion will sometimes be found to transmit a clear metallic sound (penny sound, *signe de sou*) quite unlike that heard through healthy or solid lung.

(vii.) A small protrusion, in the lateral region, distended during expiration, receding during inspiration, and due to perforation of the pleura and intercostal space, may be met with even in moderate serous effusion.

(viii.) A systolic murmur, having the characters of a pulmonary obstructive murmur, sometimes concurs with pleural effusion; disappearance of the effusion being attended by disappearance of the murmur.

C. *Puncture.*—Puncture of the chest, by means of a fine tubular needle adapted to a small exhausting syringe, is the most decisive means of determining the presence of pleural effusion. Moreover, puncture ascertains the quality of the effusion. The bare suspicion of a pleural effusion, however small it may seem to be, is a sufficient reason for exploring the chest by puncture, inasmuch as we know that to pierce the lung with a clean, fine needle is harmless.

Puncture is made where the signs of effusion are most marked; due regard being paid to the anatomy of the parts within, so as to beware of wounding the heart, diaphragm, or great vessels. But, if possible, let the puncture be made somewhere between the angle of the scapula and the edge of the pectoralis major, and not much below the nipple level. As matter of fact, the puncture is most usually made somewhere about the angle of the scapula.

Puncture may lead us into error. (i.) In small, old empyemata the enclosing walls are sometimes very thick, and it asks some faith in our power of diagnosis to let us push the needle boldly through them so as to reach the pus. (ii.) Pus is sometimes so thick that it will not pass through a fine needle: in this case a small quantity (less than a drop perhaps) will probably have entered the needle, and can be blown out and examined; if there be pus, a larger needle must be used next time. (iii.) Pus can sometimes be drawn from a bronchial tube, or from a suppurating cavity within the lung, such as produced by tubercle, destructive pneumonia, or actinomycosis. (iv.) The needle may draw off pus from the pericardium or a subphrenic abscess, after perforating the lung or the diaphragm.

D. *Different kinds of liquid effusion.*—It is by means of puncture that the kind of effusion is discovered.

1. Serous effusion.—It has been already remarked that what is called a serous effusion[1] consists of diluted liquor sanguinis. The specific gravity is usually from 1018 to 1024; but in proportion as the effusion approximates to the nature of hydrothorax the specific gravity falls, and it may be so low as 1006. Reaction, alkaline. Colour, yellowish from serum-lutein. Proteids present: fibrinogen, serum-globulin, and serum-albumin. A small quantity of sugar is often found. The liquid is seldom or never quite clear and transparent. Opalescence, when slight, is due to a few leucocytes, particles of fibrin, albuminous particles, minute oil globules, cholesterin. When the turbidity is great the effusion is called opaline or chylous, a condition which will be described further on (see p. 357). The fibrin present coagulates soon after the effusion is drawn off. The quantity of fibrin differs much in different cases; it may amount to no more than a few filaments floating in the serum, or it may be so abundant as to coagulate into a firm jelly.

2. Purulent effusion.—Pleural empyema is probably such from the first in most cases. Yet a serous effusion may possibly become purulent, a change which is either spontaneous or the result of operation. Spontaneously the change takes place slowly, a serous effusion becoming gradually purulent in the course of about three weeks. When the change is due to operation upon a serous effusion (that is to say, to infection of the effusion by septic matters), suppuration occurs more quickly, in a few days instead of weeks.

Pus is sometimes remarkably glutinous, so that, as it escapes during paracentesis, it stands in a heap when drawn into an aspiration bottle.

Pus sometimes contains much gas in solution, so that it effervesces in an aspiration bottle. Such pus is not necessarily offensive.

Pus is sometimes very fœtid, and the cause is not always the same. (i.) The cause is sometimes obscure, the pus is fœtid from the first; I have known such a case to end in recovery after a single paracentesis, without draining. (ii.) The cause is sometimes manifest; the pus becomes offensive through contamination with putrefactive microbes in such manner as the following:—gangrene of lung, perforation of lung, perforation from without (for example, an operation), perforation of diaphragm by hepatic or subphrenic abscess, mere contiguity of an offensive abdominal abscess without actual perforation. Fœtid empyema is sometimes associated with—(i.) sloughing of pleural false membranes, and even of the pleura itself (an offensive slough lying loose in the empyematous cavity may be the cause of the fœtor); (ii.) necrosis of one or more ribs.

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[1] Serum seu lympha coagulabilis, De Haen, *Ratio Med.* IV. cap. iii. p. 74.

3. Blood mingled with effusion.—Not hæmothorax, which signifies extravasation of pure blood into the pleural sac. The effusion, which is bloody, is either serous or purulent, and the proportion of blood differs much in different cases.

The conditions under which an effusion becomes bloody are these:—(i.) Simple uncomplicated pleurisy, the hæmorrhage being probably due to rupture of embryonic vessels in the false membranes; the hydrothorax of heart disease; the pleurisy of scarlatinal renal dropsy. (ii.) Acute tuberculosis of the pleura. (iii.) Cancer, sarcoma, lymphadenoma of the pleura. (iv.) Hæmorrhagic diathesis. The patient is sometimes markedly anæmic from the loss of blood, sallow, cachectic. The prognosis depends upon the cause, the fact of a bloody effusion in itself makes no difference. Cancer of the pleura is by no means the most frequent cause of bloody effusion, and the effusion in cancer is sometimes clear yellow serum.

4. Opaline serous effusion. — The effusion is opaline, milky, in consequence of abundant molecular matter suspended in it; a few leucocytes are often present, and sometimes a few red discs: these latter may be numerous enough to give a reddish colour to the effusion. Specific gravity the same as that of ordinary pleural serum. The conditions of opaline effusion are these:—

(i.) Sometimes the opacity is really chylous; for instance, if the thoracic duct be torn across, so that chyle is effused into the right pleura: in this case the molecules are all fatty, and rise to the top of the effusion, like cream. Obstruction to the duct may possibly have the same effect.

(ii.) But in most cases there is no reason for suspecting any lesion of the chylous system. The particles are often by no means all fatty, indeed very few may be fatty; they seem to be some ill-known form of proteid. Whence they come is quite uncertain; disintegration of pus globules has been supposed to be the source. Cholesterin crystals may be present, usually very few, but now and then they are very abundant, so that the opacity is chiefly due to them. No deduction can be drawn, as to the nature of the pleurisy, from the fact of the effusion being opaline.

E. *Loculated empyema*, or pleural abscess; the purulent effusion not occupying the whole of the pleural cavity, and being enclosed by adhesions, the rest of the pleural sac being natural or obliterated by adhesion.

1. The commonest seat of a circumscribed effusion is in the lateral or posterior part of the lower half of the chest on one side. In this case the diagnosis is easy enough by physical examination and puncture.

2. Loculated empyema sometimes lies between the base of the lung

and the diaphragm; mostly on one side only, but occasionally on both sides, without communicating. The diagnosis depends upon the situation of the pain felt, namely, at the attachment of the diaphragm around the lower margin of the thorax, upon immobility of the diaphragm and hypochondrium on the affected side; upon increased resistance of the hypochondrium to pressure; upon the signs of more or less extensive solidification of the base of the lung on the same side, in consequence of collapse of the lung and associated congestion (a small empyema will sometimes cause very extensive collapse, p. 368), indicated by loss of percussion tone (now and then the tone is clear and tubular, the lung being relaxed only), and weakened breath-sounds. The breathing is apt to be painful and difficult. Diaphragmatic empyema is often associated with subphrenic or hepatic abscess, and is often quite latent, found on post-mortem examination only. When a loculated empyema of this kind contains gas, diagnosis is often difficult (see page 370, Subphrenic abscess).

3. Abscess between the lobes of a lung is less common. The pus is very often discharged through the lung and expectorated, as early, it may be, as three or four weeks from the onset of the pleuritic symptoms. As a rule, it is only when the patient has begun to spit pus that the disease can even be suspected; physical signs, if any, are inadequate to the diagnosis.

4. Empyema at the apex only of the pleural cavity is an uncommon event, but one which sometimes occurs. Diagnosis is rendered all the more difficult on account of the reluctance with which we make a puncture in this dangerous region.

F. *Pulsating Empyema.*—That is to say, empyema which pulsates rhythmically with the heart.

The empyema is commonly very large, occupies and fills the left pleural cavity. (i.) The effusion usually points in one or two places, which alone pulsate. This bulging occurs in the normal heart region or in the lowest interspaces. In rare cases the protrusion has been seen in the loin below the ribs. The bulging is never larger than an orange. (ii.) Less commonly, the effusion nowhere points or bulges through the chest wall. However, in these cases also, the pulsation is usually limited to the normal heart region (to the left of the sternum), or to the lowest three or four intercostal spaces. But sometimes the pulsations are seen and felt over almost the whole of the left side.

Whether the empyema bulge or not, the heart is much displaced to the right. Pericarditis may concur, but usually the heart remains healthy. Auscultation of the pulsating part may detect conducted heart-sounds. Palpation detects no thrill and no expansion like that of an aneurysm.

Paracentesis very much helps the diagnosis. By removing part of the liquid the pulsation ceases; but the heart, being fixed by external pericardial adhesions, does not return to its normal position. The puncture needs not be made at the spot which pulsates.

The effusion is mostly chronic, and the lung wholly collapsed. Pneumothorax often concurs; in this case, pulsation is conveyed by the liquid only. The effusion is purulent in the great majority of cases, but now and then a serous effusion has been known to pulsate.

Very seldom the empyema does not fill the whole pleural cavity, but is loculated and enclosed in adhesions. This kind of pulsating empyema always bulges; it may be to the right of the sternum, but still in close neighbourhood to the heart.

The diagnosis is from intrathoracic aneurysm, and from the very uncommon condition of a pulsating cancerous tumour. Aortic aneurysm and pulsating empyema may coexist.

Pulsating empyema is, in most cases, incurable.

IV. Course and Termination. — 1. *Adhesions.* — When pleurisy terminates favourably, it is by the formation of more or less extensive adhesions between the opposed pleural surfaces, the pleural cavity being proportionally obliterated. The patient has recovered, and it is assumed, for this reason, that adhesion has occurred. Yet the recovery from pleurisy without effusion, and even from pleurisy with effusion and empyema, is often complete so far as physical signs are concerned; and the most careful examination fails to find contraction of the chest or any other signs of past disease. If adhesion be attended by physical signs, they are those which indicate unilateral contraction of the thorax and imperfect expansion of the lung. The more marked these signs, the more dense and tight may the adhesions be assumed to be.

2. *Serous effusion.*—(i.) Absorption. Serous effusion tends to be spontaneously absorbed; a large effusion may thus disappear in a week or two.

The temperature, if it have been raised, usually remains raised until absorption is complete.

The physical signs which indicate the progress of absorption are these:—The diaphragm and mediastinum go back to their natural position; to follow the retreating heart, liver, and spleen is the best means of marking the process so long as the quantity of effusion remains great. The distension of the affected side becomes less, and accurately to register this fact is an important service rendered by the cyrtometer. When the effusion has so far diminished that the lung again comes into contact with the chest wall, percussion usually enables us to follow the falling level of liquid. And, at the same time, auscultation will sometimes inform us when and where actual contact of the opposed surfaces of the pleura has occurred, friction sound being heard.

The manner in which the effusion is absorbed is not constant, but usually the liquid disappears in something like the following order:—From the vertebral groove near the root of the lung; from the supramammary region; from the rest of the vertebral groove and infrascapular region; from the inframammary region; and, lastly, from the lower lateral region, concerning which it is important to remember that the lowest part of the pleural cavity, in the erect position of the body, is in the axillary line. Thus, the upper surface of the liquid, when it reaches as high as two inches above the nipple level, is horizontal; when lower than this, the dulness forms irregular parabolic curves, which become smaller and smaller, and last of all disappear in the lowest parts of the thorax. But we must be prepared to meet with exceptions to these rules, and to find the residue of liquid in almost any part of the chest. Moreover, a large pleural effusion is sometimes absorbed, not from above downwards, according to the rule, but equally all over the side at once, friction or pleuritic râle becoming audible all over the side at once.

Disappearance of effused liquid at any spot is sometimes attended, for a day or two, by friction sound, indicative of restored contact between the pleural surfaces, redux friction as it is usually called.

Dulness, practically absolute, and due to unexpanded lung, often remains for a long time after all the effusion has been absorbed. For this reason it is often impossible to say, from physical signs alone, when the effusion has been absorbed. The physician must judge from all the signs and symptoms taken together, and especially from permanent defervescence, if the patient have been febrile. More or less dulness often remains for the rest of life.

The latest physical sign, dependent upon absorption, is retraction of the affected side. Cup-like sinking of the lower part of the sternum occasionally ensues. In some cases these deformities tend to disappear gradually, in others they are permanent.

A systolic murmur, having the characters of a pulmonary obstructive murmur, sometimes concurs with pleural effusion; disappearance of the effusion being attended by disappearance of the murmur. A permanent murmur of the same kind is sometimes heard when one side of the chest is left contracted.

(ii.) Permanence.—If the whole lung be very much reduced in size and quite inexpansible, a serous effusion will probably be permanent and endure to the end of the patient's life. It is possible that, under these circumstances, the chest walls may contract, and the mediastinum and diaphragm be displaced to such a degree as to allow of absorption of the liquid and obliteration of the pleural cavity; but these events seldom happen in the case of serous effusion. The conditions of lung which lead to its complete inexpansibility are two: carnification (see p. 368), associated with tight, unyielding thickening of the pulmonary pleura; and contracting cancer, which may reduce the whole lung to a mass not larger than the pancreas.

3. *Empyema.*—(i.) External rupture.—An empyema, left to itself, will usually perforate the thoracic wall in course of time. The opening mostly occurs in front; a common situation is the fifth interspace in the nipple line. But an empyema may point almost anywhere, from just below the collar-bone to the loin or even the buttock. The first effect of a pointing empyema in some cases is to produce what looks like a mere subcutaneous abscess; in fact an abscess of this kind over

the ribs is often due to the perforation of a pleural empyema, even if there be no signs of pleural effusion.

The course of an empyema (unless it be very small) which has been allowed to discharge spontaneously through the chest wall, and which is left to itself, is very tedious. If the opening close, it takes a long time in doing so, but often it never closes. In either case the patient runs the risk of a ruined state of health, complicated by lardaceous changes in the viscera.

(ii.) Rupture through lung.—In this case a small hole, which allows of direct communication between the empyema and a bronchial tube, is made through the lung by ulceration; or else, more seldom, the pus filters through a small portion of lung which is spongy and penetrated by many minute passages.

Empyema, which perforates the lung, is usually loculated, and often so small and deeply seated that it cannot be detected by physical examination. Such loculated empyemata often occur between the lobes of a lung, or between the lung and diaphragm, or in the mediastinum close to the root of the lung.

The expectorated pus is sometimes fœtid, sometimes not. It is sometimes fœtid at first, and afterwards spontaneously ceases to be fœtid. In some cases the opened cavity contains air, in others not. The microbe present is usually pneumococcus.

Recovery often occurs, and in no great space of time, even when the patient is left to the unassisted powers of nature, as is very often the case, it being impossible to open a deeply-seated abscess by simple paracentesis. Death may be very unexpected, the patient being choked by the sudden discharge of a large quantity of pus into the air-passages. Or death may be the termination of a long period of purulent expectoration and gradual exhaustion of the patient's strength.

(iii.) Rupture into other parts.—Empyema will sometimes perforate the pericardium, and in the case of pneumo-empyema the pericardial sac may contain air as well as pus. The peritoneum may be perforated. The empyema may discharge through the œsophagus. It is probable that the cases narrated by older physicians, cases in which empyema has been accompanied by a discharge of pus from the intestines or with the urine, were really cases of empyema complicated with subphrenic abscess.

(iv.) Incurable empyema.—Empyema is sometimes permanent and incurable because associated with certain local conditions which prevent recovery. The lung may be quite inexpansible, either carnified or tightly bound by thickened pleura. Tubercle may have invaded the lung extensively. When empyema follows upon pneumonia the pulmonary inflammation sometimes is never resolved, the lung remains hepatised, and if the patient live long enough the hepatisation will tend to pass into cirrhosis. The corresponding branch of the pulmonary artery may be closed by a thrombus. And, lastly, extensive necrosis or erosion of the ribs may ensue, in which case the pus is not necessarily offensive.

(v.) Great deformity.—More or less contraction of the affected side is an almost necessary result of a healed empyema which has occupied the whole or the greater part of the pleural cavity. When the lung is totally unexpanded the contraction will be great, the spine much curved, the mediastinum, heart, other lung, and diaphragm displaced towards the affected side. In course of time the heart will become dilated, especially the right chambers, and this is one way in which the patient may die at last from the consequences of his empyema, even although it may have closed long ago.

V. Associated diseases.—Pleurisy is often accompanied with other diseases which impede or prevent recovery.

1. *The pleura of the other side* sometimes becomes inflamed, and the patient suffers from double pleurisy. Recovery, and quick recovery, is not uncommon; and even although the case be one of double empyema, appropriate treatment will usually cure the patient.

2. *Collapse of the lung* on the other side may occur in an infant and be necessarily fatal.

3. *Gray induration* (fibrous change, cirrhosis) will sometimes ensue upon collapse of the lung. But collapse may last for many years without being followed by fibrous change, a fact proved by examination post-mortem.

4. *Gangrene* of a portion of the lung may occur when fœtid pus penetrates it from an empyema—a serious complication.

5. *Pneumothorax* is often associated with pleural effusion, and in one of two ways. Either the pneumothorax and effusion occur simultaneously, in consequence of rupture of the lung, in which case the effusion is usually purulent, but may be serous; or the pneumothorax is secondary to the pleural effusion: an empyema has opened up a bronchus by ulceration, or has discharged through the thoracic wall, or, what is more common, a pleural effusion (serous or purulent) has been removed by paracentesis, and air has passed out of the lung into the pleural cavity, not through puncture of the lung, but through rupture of it by atmospheric pressure from within.

6. *Tubercle of the lung* associated with pleurisy has been already referred to. Also the fact that many cases of pleurisy are due to tubercle of the pleura, the source of infection being, in some cases, the bronchial glands, which lie at the root of the lung covered in places by nothing but pleura. Tuberculous pleurisy is attended by exudation of organisable lymph, or serum, or pus, or by hæmorrhagic effusion. But pleural liquid effusion is sometimes concurrent with progressive pulmonary consumption, a complication which cannot be detected by physical examination until the effusion has been absorbed. Examination of the sputa for bacilli affords the only means by which pulmonary disease can be discovered during the presence of pleural effusion. When, as is sometimes the case, the phthisis is on the side opposite to that of the effusion, diagnosis is less difficult. More distant organs sometimes suffer from tubercle during the course of pleurisy, and thus

the patient's death may be hastened: the meninges of the brain are especially apt to be so affected in the young. Lastly, this seems to be a convenient place to say that a considerable proportion of persons who have recovered from pleurisy become tuberculous afterwards, and die within ten or twelve years from pulmonary consumption, or some less common form of tuberculosis.

7. *Pericarditis* often coexists whether the pleurisy affect the left side or the right. Sometimes, but seldom, perforation of the pericardium has taken place. In any case pericarditis is apt to go on to large effusion of serum or of pus. The pericardial effusion is usually not detected during life, the physical signs of that condition being hidden by those of the pleurisy. This is unfortunate, because the complication is very serious, and the patients generally die. Pneumopericardium as a result of pneumo-empyema has already been mentioned.

8. *Peritonitis* may concur. It is sometimes acute, purulent, and speedily fatal. Or it is chronic, and in this case is often tuberculous, ascites or universal adhesion being the result: the patient may recover even after his pleural effusion has been complicated by ascites. The certain diagnosis of acute and of chronic peritonitis is often impossible during life. When ascites is present, the legs are sometimes anasarcous; this condition also may end in recovery.

9. *Dilatation of the heart* sometimes follows pleurisy, especially when both pleuræ are obliterated by old adhesions, and when the lungs are imperfectly expanded. Under these conditions universal dropsy may ensue.

10. Dropsy, that is to say, anasarca and ascites, sometimes occurs even in acute pleurisy with effusion on one side only, there being no evidence of nephritis or of disease of the heart, and the patient recovering completely in about three months. In such cases the dropsy must be due to stagnation of blood in the right side of the heart.

11. *Nephritis*, indicated by the appearance of blood and tube-casts in the urine, sometimes occurs suddenly in the course of empyema under treatment by drainage. The nephritis will probably last four or six weeks and end in recovery. The cause is probably a morbid poison produced by the empyema. That pleurisy and pleural effusion are frequent complications of chronic nephritis may just be mentioned in this place.

12. *Abscess of the brain* is a consequence (not very uncommon) of empyema. The abscess is usually single, and occupies either the occipital or temporo-sphenoidal lobe: in a few rare cases many abscesses have been found. The abscess sometimes bursts into the lateral ventricle; and in this way even the subarachnoid space of the spinal cord may become filled with pus. This cerebral abscess is probably metastatic, and due to the transportation of a microbic embolus from the thoracic disease; but any other signs of pyæmia are seldom observed either before or after death: why the white matter of the brain alone should be selected for embolism is unknown. The onset of

cerebral abscess is very insidious; for a long time the only symptom is headache of varying severity, sometimes little, sometimes much: so far as any distinctive symptoms go, the disease is latent. Towards the end, a few days or a week before death, much more decisive signs of disease are superadded to the headache; namely, vomiting, optic neuritis, general convulsions, coma. Or, as sometimes happens, the patient dies very unexpectedly, without the occurrence of any grave warning symptoms.

13. *Hemiplegia* due to softening of the brain is another possible consequence of empyema. No doubt the softening is sometimes caused by embolism of the middle cerebral artery; the embolus being derived from a thrombus which has formed in the heart or pulmonary veins during the stagnation of the circulation which is a necessary result of compression of the lung and displacement of the heart. Sometimes hemiplegia occurs during or soon after paracentesis, a thrombus or a portion thereof being dislodged during the commotion of parts which must follow upon removal of much liquid. In rare cases this hemiplegia is temporary, and the patient recovers in a few hours or days. But the softening of the brain which causes hemiplegia is not always to be explained by embolism; it may be that no arterial lesions of any kind are to be found after death; and a local metastatic encephalitis, not going on to suppuration, seems to afford the most probable explanation (6*a*). Other symptoms may depend upon the softening, according to its locality; namely, aphasia and associated defects; amaurosis, with a natural condition of the retina. Or the softening may involve both sides of the brain, with the consequences of general paralysis and dementia.

14. *Lardaceous disease* is a consequence which nowadays is seldom met with. In this case the empyema is usually chronic and fistulous; but even a small empyema which has never been discharged may be attended by this form of degeneration.

15. *Clubbing of the finger-ends* attracted much attention from the ancient physicians. The symptom may be well marked at the end of a fortnight from the beginning of an empyema. Clubbing will sometimes disappear gradually when empyema has been cured.

SAMUEL GEE.

Morbid anatomy.—The pleural affection does not necessarily vary with its exciting cause. Pleurisy, whether primary or secondary, may present the same appearances both to the naked eye and to the microscope. As in all serous inflammation, several factors are present: hyperæmia, proliferation, and desquamation of the endothelium, proliferation of the sub-endothelial connective tissue cells, exudation of fluid, and escape of leucocytes from the blood-vessels into the cavity, the formation and deposition of lymph on the surface, and finally the organisation of the lymph into fibrous membrane or adhesions. The difference in different cases consists principally in the amount of fluid and in the proportion of fibrin and leucocytes which it contains; but on the one hand the same exciting cause may produce in one case a "dry" pleurisy, in another a serous, and in a third a purulent effusion; and on the other hand these various forms may pass imperceptibly from one into another. The driest pleurisy is attended with some fluid exudation, and the clearest pleural effusion contains some fibrin and some leucocytes. These are, therefore, but stages in one process.

At the onset of pleurisy the surface of the membrane can just be seen to have lost its polish; and if the inflammation be more advanced, the fingers also can feel a slight roughness. This is due both to endothelial proliferation and to fibrinous deposit. If the disease go no farther, these products may disappear, and the membrane show no sign of the attack. But probably, in all cases which reach beyond the very slightest degree, both the parietal and visceral surfaces become affected, follow the plastic tendency of all serous membranes, and eventually form adhesions with one another. The extent to which this takes place varies from the production of a few fibrous threads to general adhesion of the whole apposed surfaces.

If the inflammation be more intense, there is exudation of fluid containing some fibrinous shreds. It varies from a few drachms up to an amount sufficient to distend the chest and displace the viscera. The amount of fibrin in it also varies greatly; though produced wherever the pleura is inflamed the fluid tends to collect at the lowest part. Occasionally this tendency is counteracted by adhesions, so that a fluid collection is limited to some other part of the chest than the base. Where it lies it takes the place of the lung, which, thus relieved from the suction of the chest wall, collapses beneath the fluid. When the exudation is large enough to exert positive pressure in the thorax, the lung is forcibly compressed also. While the fluid is effused, layers of lymph may at the same time be formed upon the pleura, and the membrane thus formed may so swathe the lung that inspiration has not force enough to expand it as the fluid is removed.

An originally serous effusion may become purulent; but the great majority of purulent effusions are probably purulent from the first. The fluid in these cases varies greatly. It is sometimes liquid and laudable with very little fibrin; in others, and especially in chronic cases, the fibrin may form large curdy masses; in others, again, and especially in those of a septic nature, the fluid is much thinner than pus. The purulent or puriform effusion is usually inodorous; but it may become putrid when, by a wound of the chest or through the lung, access has been given to the open air; or when, as occasionally happens, the pleura communicates with an abdominal abscess or with the alimentary tract.

Lastly, the effusion may be bloody or may be almost pure blood.

Pathogeny.—The exciting causes of pleurisy are manifold. Those cases which are secondary to heart disease are, so far as we know, mechanical in origin. Pleurisy occurring in the course of nephritis may be of the same nature (2). But evidence is accumulating that under other conditions pleurisy is directly due to microbes. Purulent effusions were the first to be studied from this point of view. Ehrlich found micrococci in three cases of puerperal septicæmia with empyema. Rosenbach, Hoffa, and Weichselbaum also verified their presence in all the cases of empyema, eleven in number, which they examined. Kracht confirmed this in ten cases. Fränkel examined twelve cases; in three cases of primary pleurisy he found the streptococcus pyogenes; in three the diplococcus pneumoniæ; in four which were of tuberculous origin he discovered the tubercle bacillus in one alone, the others giving negative results; and in two cases secondary to other abscesses he again found the streptococcus. Meanwhile the influence of tubercle bacilli in the causation of pleural effusions, whether serous or purulent, on which great stress had been laid by Landouzy from the clinical point of view, had been studied by Kelsch and Vaillard, Gombault and Chauffard, and Gilbert and Lion. They were not very successful. Kelsch and Vaillard, inoculating in animals, could only reproduce tubercle from two out of four empyemas, and from one out of ten serous effusions. Gombault and Chauffard failed nine times and succeeded eight times. Gilbert and Lion failed altogether in twenty cases. Levy examined fifty-four cases, of which thirty-seven were serous, seventeen purulent effusions; six were secondary to typhoid fever, of which three contained staphylococcus pyogenes, and three were negative; nineteen were secondary to pneumonia, broncho-pneumonia, or influenza, of which three were negative; fourteen revealed diplococcus pneumoniæ, and two contained the staphylococcus; in one of these also the diplococcus was found. In fourteen tuberculous cases Levy failed to find the tubercle bacillus. In one case secondary to rheumatism, and in seven secondary to heart disease, nephritis, and cancer, the result was negative; but in one hæmorrhagic effusion secondary to infarct of the lung and in six other mixed cases the staphylococcus was present. Renvers and Prince Ludwig Ferdinand confirmed these results. Pansini in fifteen serous effusions had five negative results, but found tubercle bacilli six times, diplococcus thrice, and streptococcus or diplococcus once; in eight empyemas he found tubercle bacilli thrice; and in all but one of the rest the strepto-, staphylo-, or diplococcus. In one sanguineous effusion he found the tubercle bacillus. Netter (13) examined 109 cases of empyema: the diplococcus was present twenty-nine times alone, thrice with streptococcus; streptococcus was found alone in forty-eight cases, and staphylococci in two cases. Of fifteen cases of fœtid effusion saprophytic organisms were found in all, and of twelve tuberculous cases the tubercle bacillus was present in six. He points out the much greater benignity of the diplococcus, and explains by this fact the more frequent recovery of children; for of twenty-eight cases in children the diplococcus was present alone or with the other two cocci in fifteen—a rate of 53 per cent, which is exactly that of the streptococcus in adults. In a second paper (14) he stated that he had been able to produce tubercle by inoculating guinea-pigs with the serous effusion drawn off by a Pravaz syringe in seven out of twelve cases which could be diagnosed clinically as tuberculous, and in eight out of twenty cases of "idiopathic" pleurisy. Koplik gives confirmatory evidence of cases in children. Sacaze found tubercle bacilli at the beginning of a serous effusion, but failed to produce it later; which result throws some light on the difficulty always encountered in showing its presence, even when clinical evidence of a tuberculous process is strong. Hanot discovered the bacillus in a hæmorrhagic effusion (7) (21).

Both serous and purulent effusions, when primary, are therefore due to the three micrococci mentioned, and to the tubercle bacillus; and this so frequently, that as observers become more skilled this rule will probably be found universal. More than one of the above authors venture to state that where micrococci are not present the case will almost always prove to be tuberculous. Hæmorrhagic effusions when not due to cancer or to some rarer cause are also probably tuberculous.

The pathology of rheumatic pleurisy is as yet unknown; and the same may be said of the pleurisy which French authors (1) describe as occurring in the secondary stage of syphilis.

W. P. HERRINGHAM.

VI. Diagnosis.—Pleurisy is simulated by certain other diseases in respect either of symptoms or of physical signs.

1. The pain of *pleurodynia* is, by itself, indistinguishable from that of pleurisy. Diagnosis becomes possible when there are other signs or symptoms of pleurisy; for pleurodynia is mere pain, and pleurisy is sometimes indicated by pain alone.

2. The *rale* (not friction sound) which in rare cases attends the onset of pleurisy closely resembles the rale of bronchitis (see p. 352). The difference lies mainly in this, that the rale of pleurisy tends to be heard over one side only of the chest, and the rale of bronchitis over both sides. Pleuritic rale is soon superseded by other signs of pleurisy.

3. *Acute collapse* of extensive portions of lung is a condition which is very apt to occur in young children as a result of obstruction to a bronchial tube. The case of obstruction by an inhaled foreign body need not be considered here, for the whole course and symptoms of this accident are not at all like those of pleurisy with effusion. But bronchial catarrh, and even slight bronchial catarrh, will sometimes cause extensive collapse in a young child, or in a very feeble patient who is not a young child. Bronchitis setting in suddenly, with fever, cough, tightness of the chest, vomiting, and followed in a day or two by the signs of collapse at the lower part of one lung (namely, dulness to percussion and weak breathing), counterfeits pleurisy with effusion very closely. Diagnosis may be impossible at first. Usually the catarrhal infarct soon clears up; if it be deemed necessary a puncture may be made.

4. *Chronic collapse of lung and Cirrhosis* are two conditions which closely resemble each other in the living subject, and which often cannot be distinguished excepting upon the post-mortem table. Nor is it of any practical importance that they should be distinguished; the

useful term carnification (invented by Laennec) may be taken to include them both. The physical signs of carnification of the lower lobe of a lung and those of a small pleural effusion are the same, excepting that the chest may be distended on one side, and the heart be displaced away from the disease in some cases of local pleural effusion. But now and then the chest is contracted and the heart not displaced even in a pleural effusion. The symptoms afford no help to diagnosis, and the right understanding of a case may be rendered all the more difficult by the fact that carnification is not only a constant result of pleural effusion, but often persists long after the effusion has disappeared (see p. 360). The chief means of distinguishing between the two conditions is puncture. Yet, under these circumstances, puncture sometimes fails to detect pleural effusion, and chiefly for this reason, that carnification is often much more extensive than the effusion which causes it. For instance, a small pleural effusion, lying upon the diaphragm or in the posterior mediastinum, will sometimes be attended by collapse of the whole lower lobe of a lung; and this carnified lung being the only portion of disease which is in contact with the chest walls, the physical signs will be wholly dependent upon the carnification, and if puncture be made it cannot hit the effusion unless the needle go right through the lung. Wherefore it may be impossible to say whether there be an effusion or a mere carnification. Sometimes the expectoration of a small empyema occurs so as to clear up our doubts.

5. *Tuberculous phthisis* of a lower lobe resembles a small pleural effusion in many respects which it seems hardly necessary to enumerate; puncture and microscopic examination of the sputa are the most trustworthy means of distinction. But there is an especial form of pleurisy which, for a time, is indistinguishable from pulmonary tuberculosis. In this case the pleurisy involves the whole of one side, which is retracted, it may be considerably, and moves much less freely than in health. The percussion note is raised in pitch and muffled over the greater part or the whole of the side; the sense of resistance is increased; when the disease affects the left side the superficial area of cardiac dulness is extended. The respiration generally is weak, and attended by friction sound at some part, or by widespread rale indistinguishable from the mucous rale of catarrh or phthisis (see p. 352). At places the breath-sound may be bronchial, in all degrees of intensity, up to perfect cavernous resonance. Add to these signs hectic fever with diarrhœa and vomiting, and it is easy to understand why pleurisy of this kind is apt to be mistaken for phthisis more or less advanced. The pleurisy terminates in one of two ways. Either the physical signs of disease gradually disappear, excepting perhaps that a slight unilateral retraction of the chest, or a cup-like depression of the sternum, is left behind, the patient recovering at the same time his former state of health; or signs of a small effusion slowly appear at the base, and, when the chest is punctured, a little pus is withdrawn and the case comes into the category of empyema. Whenever the signs of a case of supposed

phthisis are in some respects peculiar; whenever they indicate advanced and extensive disease, but limited to one side of the chest; whenever cavernous signs are heard in unusual places;—it is well to weigh the possibility of simple pleurisy, and not to rest confidently in the diagnosis of phthisis until tubercle bacilli have been found in the sputa.

6. *Acute pneumonia* is seldom mistaken for pleural effusion unless the tubes of the pneumonic lung be so plugged with mucus that conduction of the breath-sounds is obstructed. It much more often happens that a small pleural effusion is mistaken for pneumonia. The physical signs of the two diseases may be the same, and even puncture is not always decisive; should an empyema be confined to the apex of a pleural cavity, so infrequent an occurrence, compared with the frequency of apex pneumonia, will render diagnosis unusually difficult (see p. 358). The symptoms of the two diseases may be the same, especially in the pleurisy which is due to pneumococcus (see p. 352); not seldom in this case the patient dies before certain diagnosis becomes possible: a physician well read in the book of nature knows that he cannot always distinguish between pleurisy and pneumonia.

Chronic pneumonia—that is to say, hepatisation slow to resolve—will resemble in many respects pleural effusion supervening upon pneumonia.

7. *Malignant tumour* of the lung closely resembles pleural effusion in respect of the physical signs. A tumour does not often cause enlargement of the affected side, or displace any organs, yet now and then a quickly growing tumour will produce these effects. When dulness begins not at the bottom of the chest; when there is a great extent of absolute dulness in front and none behind; when, in the midst of a great extent of dulness, we detect one or more small insulated patches of resonance (perhaps quite clear or even cracked-pot), we may debate the existence of solid tumour. The crucial test is puncture.

A large serous effusion (see p. 362) is sometimes the necessary result of contracting cancer of the lung. The nature of the case may be suspected if cancer can be discovered elsewhere, and especially if large hard glands can be felt above the collar-bone or in the armpit.

8. *A large hydatid cyst* will yield most of the signs of pleural effusion; namely, unilateral distension of the chest, displacement of the diaphragm and mediastinum, dulness to percussion, and weak or absent breathing sound. An exploratory puncture is the most decisive means of diagnosis; the fluid of hydatid being free from albumin and more watery than that of pleural effusion, to say nothing of the possible discovery of echinococcus hooks. But if the hydatid have suppurated, the nature of the disease is sometimes not suspected until a free opening has been made, such as to permit the escape of hydatid membrane. (For full discussion of Thoracic Hydatid, *vide* vol. ii. p. 1137.)

9. *Actinomycosis* of the base of the lung simulates pleurisy with effusion, and is, indeed, sometimes attended therewith. The diagnosis

cannot be made until the fungus is discovered in the sputum, or until the growth perforates the wall of the chest (*vide* vol. ii. p. 81).

10. *Subphrenic abscess* is much more common on the right side than on the left, for reasons which become clear when the antecedents of the abscess are considered. It is often, if not always, associated with pleurisy on the same side, and usually with empyema, due to perforation of the diaphragm or not. Hence empyema on the right side in a person who has probably suffered from tropical hepatitis, from simple or cancerous ulcer of the stomach, or from other causes of subphrenic abscess, should always lead us to reflect upon the possible coexistence of this disease. The pus of subphrenic abscess and of the empyema is fœtid. The abscess, even if there be no empyema, may burst into the lung, and lead to expectoration of most offensive pus. Whether there be an associated thoracic empyema or not makes little difference so far as the physical signs of a subphrenic abscess are concerned; for the empyema is local, enclosed in adhesions, and not nearly filling the pleural cavity. The signs are both abdominal and thoracic, sometimes more the one, sometimes the other. The abdominal signs are: (*a*) fulness and tightness in the hypochondrium; (*b*) the liver depressed, sometimes, but by no means always; moreover the liver is sometimes much depressed in uncomplicated thoracic empyema. The thoracic signs are: (α) dulness to percussion and signs of pleural effusion at the base, whether there be a pleural effusion or not; in the latter case the diaphragm is much pushed upwards, and the lung proportionally collapsed: (β) the heart's apex beat is sometimes displaced even in subphrenic abscess without empyema, but more often is not displaced. Puncture, made as for pleural effusion, will probably reveal the presence of pus, but will not tell us whether the pus is above the diaphragm or below it. Uncomplicated subphrenic abscess may be mistaken for simple thoracic empyema, even after the abscess has been emptied of pus by aspiration; the needle having gone right through the diaphragm, which has been pushed much upwards, as high it may be as the third rib. Even resection of a portion of a rib, and exploration of the pus cavity by the finger, do not always enable us to say at first whether we have opened a cavity above or below the diaphragm; or, in the former case, whether the diaphragm be perforated or not.

Subphrenic abscess often contains gas derived from perforation of the alimentary canal or from decomposition. In this case the disease is apt to escape discovery by physical examination, because there is no dulness to percussion. Sometimes the percussion note is clearer than natural; and sometimes the clear note is more extensive also, so that the liver dulness disappears. The resonance may possess amphoric quality. Auscultation usually detects one or more signs of a large cavity containing air; namely, amphoric hum (attending the sounds of breathing, speaking, and of the heart), metallic tinkle, bell sound, and succussion splash. If the diaphragm be perforated, the empyema will be a pyopneumothorax.

11. It is sometimes hard to decide whether *friction sound* heard over the heart region be *pleural or pericardial.* Pleural friction may be produced by movement of the heart alone; as pericardial friction may be under the influence of breathing movements.

12. Large effusion into the left pleura may cause bulging of the chest in the heart region, such as to raise the question of *concurrent pericardial effusion;* for the two diseases are often associated (see p. 369). The diagnosis depends mainly upon the result of emptying the left pleura by paracentesis, whereby alone can the signs of pericardial effusion become manifest. When a rib has been resected in the treatment of empyema, the finger passed into the pleural cavity may possibly be able to feel a bulging pericardial sac.

13. When pericardial effusion is attended by extensive collapse of lung, and the chest is punctured with a view to determine the cause of the dulness, the needle may go right through the lung and discharge liquid from the pericardium; and, until examination post-mortem, the physician may rest in the unshaken belief that the liquid came from the pleura.

14. The manner in which pulsating empyema counterfeits *aneurysm* has been already referred to (p. 358).

15. *An abscess in the thoracic walls* may be the only evidence of a small empyema (see p. 360) which has penetrated an intercostal space. Even when the abscess has been opened it is not always easy to say whether it communicates with the pleural cavity or not. It is possible that pleurisy may be the cause of abscess in the thoracic walls without actual perforation of the pleura. But more commonly parietal abscess (as distinguished from pointing empyema) is due to such causes as injury, pyæmia, periostitis of a rib, or necrosis of the same; and this "peripleuritis" may perhaps sometimes set up pleurisy. Lastly, in all cases of superficial abscess the question of actinomycosis must be pondered.

VII. Prognosis.—It seems to be unnecessary to reiterate many prognostics, which will be found in their appropriate places in the foregoing and following pages. But one fact of great importance demands special attention, namely, the occurrence of unexpected and speedy death in cases of pleural effusion. The conditions of this unexpected death are not always the same.

(i.) The sudden rupture of an empyema (and it may be quite a small empyema) into the lung is sometimes sufficient to suffocate the patient in a few minutes.

(ii.) Much more often the death occurs apart from any discharge of the effusion. The effusion is usually large, filling up the whole or greater part of the pleural cavity. The effusion is usually serous. Whether it be on the right side or on the left makes no difference. Suddenly, and often after a little exertion, the patient is seized with dyspnœa or faintness, or both. The lipothymial symptoms soon predominate; the skin becomes cold and clammy or sweating, the face and lips assume the wan, dusky, livid colour of a dying person, the pulse is small and irregular; death ensues within half an hour or an hour. The explanation of the speedy death is mostly found post-mortem in thrombosis of the right side of the heart, consequent upon stagnation of the circulation through it, dependent upon the collapsed state of the lung. This heart thrombus has one of two results: either the thrombus is propagated into the pulmonary artery, and thence into that branch of it which supplies the unaffected lung; or an embolus, derived from the heart thrombus, is driven into the pulmonary artery, or a large branch of it. But thrombosis meet to explain the death is not always found: sometimes a latent pericardial effusion is present; but sometimes nothing sufficient can be found, and in cases of this kind hypothetical explanations have been offered, such as twisting of the large vessels at the root of the heart, bending of the inferior vena cava at an acute angle, compression of one auricle of the heart, degenerative changes in the muscular tissue of the heart.

(iii.) Frothy serous expectoration sometimes suffocates the patient during or soon after paracentesis of the chest (see p. 376); or, in very rare cases, may even supervene upon large effusions apart from paracentesis.

VIII. Treatment.—A. In the treatment of pleurisy with no liquid effusion, the main indication special to the disease is to relieve pain. The most effectual means of doing so are two: subcutaneous injection of morphia at the seat of pain, or the application of a few leeches. In many cases much less decisive means are sufficient: warmth by hot-water fomentations or linseed-meal poultices; a mustard poultice, or a turpentine fomentation.

B. The treatment of pleurisy with effusion relates almost wholly to removal of the effusion.

When the effusion is believed to be recent, not large, and not purulent, it is best to defer operation for a week or two, so as to see whether the liquid can be removed spontaneously without operation. It is probable that absorption may be assisted by sundry means: iodide of potassium in moderate doses should be given; the affected side of the chest should be painted with tincture of iodine two or three times a day; blisters, the size of the palm of the hand, or less according to the size of the patient, may be employed, one blister at a time, and the sore allowed to heal as soon as possible. In the case of children blisters should not be used.

But the question of paracentesis is always foremost in the mind, and may be discussed under four heads: when, where, and how the operation should be performed, and, lastly, certain dangers which sometimes attend the operation. The age of the patient is never taken into consideration. I have treated successfully by paracentesis patients three months old and eighty-seven years old.

I. *When should paracentesis be performed?*—The answer to this question depends upon the quality of the effusion.

1. Pus must be removed as soon as possible. If it be bloody, or if the pleura contain air as well as pus, the same rule holds good. Free evacuation of pus may be expected to bring the patient's temperature down nearly or quite to the normal; if this be not the result, we may assume that there is some retention of pus. Any subsequent rise of temperature, after a fall to the normal, will most likely be due to imperfect drainage. But perfect drainage cannot always be attained, especially when a small quantity of pus is secreted in an inaccessible cavity shut off from the rest; in cases of this kind time usually surmounts the difficulty, the retaining lymph breaking down under persistent drainage.

2. Serum should be removed by paracentesis in all cases which present an effusion so great as to fill the pleura, or which are attended by any distress of breathing, or which show no signs of being absorbed after a week or ten days of the other treatment already described.

II. *Where should paracentesis be performed?*—1. When the effusion is small the puncture must be made where the effusion is believed to be.

2. When the effusion is great, so that the pleural cavity is full or almost full, the best place for puncture is in the middle line of the axillary region, about the horizontal level of the nipple or a little below it, where the intercostal spaces are wide and the muscular integuments thin. Another part of the chest which is often chosen for puncture is a spot just below the angle of the scapula, but the lung is sometimes adherent to the chest wall here, and will therefore be pierced by paracentesis; in this case pneumothorax is apt to ensue, and, what is a result far worse, but less frequent, sloughing of the perforated lung. Probably no part of the chest can be chosen as being entirely free from the risk that paracentesis may perforate collapsed and adherent lung, but the risk is less at the spot first recommended for the place of puncture than at any other situation.

III. *How should paracentesis be performed?*—1. Serous effusion should be removed by means of a trocar and canula. Whether suction be employed or not is, in most cases, a matter of no great consequence. If suction be not employed, a canula connected with a long india-rubber tube should be used, the free end of the tube being kept under liquid, so that no air can enter the chest. On the whole, suction is to be preferred, for in this way small obstacles due to fragments of lymph floating in the serum can be overcome. It is best to make no more vacuum than is necessary to maintain a gentle flow of liquid. As much liquid is to be drawn off as possible without causing any serious discomfort to the patient. Suction is to be stopped if the flowing fluid become bloody, if the patient feel much pain in his chest, or if he begin to cough much; in which last case there is the risk of serous expectoration (p. 376).

The pain of puncture is diminished if the skin be previously frozen by ice, or by an ether or chlorethyl spray.

It happens sometimes, but not often, that the most powerful suction can extract no more than a small quantity of the effusion. The usual cause of this difficulty is found in a fragment of lymph which blocks the canula or obstructs its orifice. But sometimes, even when the effusion is free from floating lymph, it is impossible to evacuate the chest. Cases of this latter kind, which are uncommon, are probably to be explained by a lung rendered inexpansible by thickened pleura or by obstructed air-tubes. Nothing more can be done than to draw off as much serum as possible, and to repeat the paracentesis in a day or two.

Very often a single paracentesis cures the patient, the little liquid left being soon absorbed. But sometimes the effusion returns, and the rule of practice is to repeat the operation as often as seems necessary. In rare cases an abundant effusion will continue for an indefinite time, but even then the only treatment is paracentesis repeated as often as necessary. Drainage by a permanent opening is out of the question, and would be certain to convert the serous effusion into empyema, to the great danger of the patient's life.

There is no reason for fear lest paracentesis alone and without drainage should convert serous effusion into pus, provided that all the instruments used be surgically clean.

2. Empyema is to be treated by incision and drainage.

(i.) When the quantity of pus is not very large it is best to make a permanent opening and drain at once. In some cases thorough and speedy drainage cannot be obtained unless a large opening is made by excising a portion of one of the ribs; and, therefore, to avoid all doubt upon this point it is good practice to resect a rib in all cases.

(ii.) When the empyema fills the pleural cavity it is safer to remove as much of the pus as possible by paracentesis at first, and to make the incision a day or two afterwards. The sudden discharge of a very large quantity of pus from the chest causes a great shock to some patients, and previous paracentesis lessens the shock. Paracentesis, and sometimes even a single paracentesis, can cure empyema. I have known a single paracentesis cure a stinking empyema of considerable size. And I have known paracentesis, which removed more than five pints of pus from the pleura, to be followed within a week or two by effusion of clear serum to the same amount in the same pleura. But cases of this sort are very uncommon, and incision and drainage are the proper treatment of empyema.

In order, then, that the drainage may be thorough it is best to remove a small portion of one of the ribs. Incision is made right down upon the rib, the periosteum is separated all round by an elevator, and the rib is divided in two places by cutting forceps, so that about an inch can be removed. It is not good practice to swill the empyematous cavity out; nothing is gained by removing false membranes in this way; a fœtid empyema is soon deodorised by thorough drainage and careful antiseptic dressing, and even if not, washing out does not help. Moreover, injections are dangerous if there be an ulcerous opening through which they can enter the lung; the shock which immediately ensues upon such an entrance puts life in jeopardy. Even if there be no such ulcer, injections are dangerous. I have known a patient die very suddenly during injection, when but a very small quantity of a weak carbolic acid solution had been injected; no chloroform was given, and nothing could be found post-mortem to explain the death. But sudden syncope coming on in this way is not always fatal. The cause of the syncope is not understood; a case has been recorded in which the right chambers of the heart were found post-mortem to be distended with gas. The syncope is sometimes followed by convulsions and coma; in this case death usually ensues within twenty-four hours. If the temperature rise much above 105° recovery is very unlikely. Yet recovery even after convulsions may occur; temporary palsy of a limb or of one side of the body has been noted upon cessation of the convulsions. In other cases the sudden syncope has been attended by palsy without convulsions, by spastic rigidity of a limb or of the jaw, or by aphasia; these symptoms commonly pass away in an hour or less. Convulsions and paralyses of this kind are probably epileptoid in nature, and quite different from the paralyses which will be spoken of hereafter, and which are due to embolism.

If there be any probability of the coexistence of pulmonary tubercle the line of treatment is not so clear. To release the lung from compression may accelerate the infective and destructive changes going on therein; to say nothing of the debilitating effect of a free purulent discharge, which there is but small chance of stopping. Under these circumstances it is best to resort to paracentesis several times at least, the result being watched before proceeding to drainage.

In dealing with small deeply-seated empyema, such as that which so often leads to fœtid expectoration, it is sometimes necessary to remove portions of two or three ribs, so that adhesions can be broken down and the cavity opened by the finger. To cut through the lung in such cases many cost the patient his life.

For a day or two after opening the chest the discharge will probably continue to be abundant. It will then, in most cases, gradually lessen until it ceases altogether in a few weeks, three or more. In proportion as the discharge becomes scantier the drainage-tube must be shortened, so as to allow the sinus to heal from the bottom.

The temperature ought to fall almost or quite to the normal after the pus has been discharged. Should the temperature remain raised, there must be either retention of pus or some other concomitant disease. When, after the fever has ceased, the temperature rises again there is probably retention of pus, and should the temperature not fall again in a few days the sinus should be probed, and a longer piece of tube be inserted if necessary. But the temperature will often rise for a few days without obvious retention of pus, and will fall again without obvious increase in the amount of discharge.

When the sinus shows no tendency to close it is best to wait two or three months before undertaking any further operation. But when the discharge continues for a longer time (and these remarks apply also to cases of neglected empyema which has been allowed to open spontaneously), and it seems necessary that something else should be done (especially when the discharge remains abundant and the health of the patient suffers), a more extensive operation must be performed. Longer portions (two or three inches) of three, four, or more ribs in the neighbourhood of the sinus must be resected, so as to allow the chest walls to fall in and meet the lung. The cases in which the discharge is not finally arrested by this operation are very few.

IV. *Dangers of paracentesis.*—1. Serous (or albuminous) expectoration. Paracentesis, by suction, of a pleural effusion is sometimes followed by expectoration of blood-serum. If a patient begin to cough much during the operation it must be stopped at once, and the patient be carefully watched. It is very probable that a small amount of serous expectoration under these circumstances is not uncommon; it is only when the secretion is abundant that the condition is dangerous and apt to end in speedy death. Serous expectoration mostly ensues during or directly after the operation, but sometimes an hour or two will elapse before the secretion becomes dangerously abundant; the latter cases are less serious. When abundant serous expectoration follows rapidly upon paracentesis the patient may die suffocated within half an hour. The stuff expectorated is frothy, viscid, transparent, neutral, or alkaline, yellow or yellowish green, with a specific gravity of about 1020, and rendered almost solid by heat and a drop or two of acetic acid. Chemically the sputum consists of serum-albumin and a little mucin. On standing there falls a scanty deposit of pus and blood corpuscles. Post-mortem the lung is œdematous, and usually fully expanded. Concomitant disease, such as disease of the heart, mediastinal tumour, or hæmoptoic infarcts, favours the occurrence of serous expectoration.

2. Pneumothorax sometimes follows withdrawal of a pleural effusion. The cause is not always the same. (i.) In some cases the lung has been injured by the operation, an accident especially apt to occur when collapsed lung, undiscoverable by physical examination, is adherent to the chest wall, so that the trocar goes through the lung. (ii.) Sometimes the air comes from a spontaneous rupture of the lung; softened tubercle may give way; in empyema there is sometimes a small ulcerous breach in the surface of the lung existing before the operation, or merely collapsed lung may burst in some small spot in the process of expansion during paracentesis by suction. (iii.) The pus of empyema sometimes contains so much gas dissolved in it that in some cases this is a very probable cause of pneumothorax.

3. Hæmorrhage from the pleuritic membranes is sometimes the result of paracentesis. If the blood flow at all freely, the operation must be stopped, and it is seldom that any bad consequence follows. But death has been due to this cause, the pleural cavity being found post-mortem to contain a large quantity of blood.

4. Fatal hæmoptysis has ensued upon evacuation of empyema in

pulmonary phthisis, which has gone on to the formation of cavity containing a small aneurysm.

5. Embolism of distant arteries, by coagula dislodged from the pulmonary veins, may be the result of paracentesis. The most common result is hemiplegia (see p. 364), which is usually incomplete. Embolism of both iliac arteries has been known to occur.

6. Sudden death has followed soon after paracentesis in rare cases, even when the pleura has not been washed out (see p. 376). In one case of this kind the right side of the heart was found to be filled by a clot, formed in all probability during life. In other cases no satisfactory explanation of the death has been forthcoming; all operations involve the possibility of the patient dying suddenly.

The treatment of fœtid expectoration from a small deeply-seated empyema, which cannot be laid open by an operation, is the same as that of a similar condition in phthisis.

Subsequent deformity.—Not much can be done to expand the collapsed lung, and to counteract the deformity of the thorax and spine which is apt to follow upon chronic pleurisy. Exercises for the arms should be prescribed, especially such as tend to open the chest in front; for instance, drawing the body up by the arms clinging to a horizontal bar, skipping backwards, the use of a chest-expander behind the back, or of dumb-bells and appropriate drilling.

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PNEUMOTHORAX

Hydropneumothorax ; Pyopneumothorax

Definition.—By pneumothorax is meant the presence of air in the pleural sac. Generally speaking, the air or gas is accompanied by serous fluid or pus ; hence the synonyms hydro- or pyo-pneumothorax to denote one or other of these composite conditions.

Etiology.—Although causes leading to the production of pneumothorax are fairly numerous, most of them, as detailed by various observers, are of remarkably infrequent occurrence; indeed the disease itself may be said to be rather uncommon.

The oft-quoted statistics of Saussier (12) give the relative frequency of the causes in 131 cases as follows :—

Phthisis	81	Fistula between pleura, liver, and intestine	2
Empyema	29	Abscess of lung	1
Gangrene of lung	7	Cancer of lung	1
Emphysema of lung	5	Hæmothorax	1
Apoplexy of lung	3		
Hydatids	1		

Even this considerable list is by no means complete, and several additions have to be made—most of them, however, of rare occurrence. For example, pneumothorax may be brought about by external injury—such as a perforating wound of the wall of the chest, by the wounding of the visceral pleura by a fractured rib, or even by heavy blows or falls, apart from wound or fracture. Indeed, cases of pneumothorax occur from time to time of which no cause is discoverable (6).

Internal injury also must be credited with the production of some cases of the disease, as when a bougie, in its passage through a cancerous œsophageal stricture, has perforated the diseased wall of the tube and entered the pleural sac. Ulceration of a bronchial tube, however produced, is another possible cause ; perforation of the diaphragm, brought about by suppuration resulting from a perforated gastric ulcer, another; and finally, cases occur in persons, otherwise apparently in robust health, as a result of strain, that is, of strenuous muscular effort with the glottis closed. All these varieties of causes group themselves in two divisions : the one containing those in which the perforation causing pneumothorax results from injury or disease directly affecting the lungs themselves, or the bronchia (and this is the more important) ; and the other containing those in which the causes of perforation are external to the pleura. An additional variety is attributed by some authors to the gaseous decomposition of liquids, such as pus, pathologically present in the pleura : this, if it ever occur at all, is infinitely rare ; there is good reason to doubt whether it does occur.

All observers are agreed that pulmonary tuberculosis, producing perforation by ulceration of the visceral pleura, is by far the most frequent cause of pneumothorax; and most of them place the proportion of such cases at about 90 per cent of the whole. The relative frequency of the disease in cases of phthisis is variously stated by different authorities as being from 3 to 14 per cent (4). My own experience would suggest the smaller number as being nearer the general average. It was found present twice only in 60 post-mortem examinations made on cases of phthisis at the Middlesex Hospital in the years 1877, 1878, and 1879.

It should be added that men are more apt to be attacked by pneumothorax than women; and it is a disease especially of the earlier periods of adult life. This latter is to be expected from its connection with pulmonary tuberculosis.

Pathology and Morbid anatomy.—The mode in which air gains access to the pleura in such cases as those of external injury, or the bursting of an empyema, are so obvious as to require no explanation. With regard to the tuberculous cases, which, as we have seen, form an enormous majority of the whole, it is in the acute forms that pneumothorax is most apt to occur. Those in which the disease of the lung progresses slowly are comparatively little likely to perforate, owing to the formation of protecting adhesions between the visceral and parietal layers of the pleura. In the former class of cases tuberculous masses become softened, and break down close under the surface of the lung; necrosis of the overlying portion of the pleura takes place; and some effort, or an attack of coughing, is sufficient to determine a rupture, or it may occur without any apparent exciting cause.

In connection with this portion of the subject it is interesting to note how the conservative processes of nature tend to the prevention of pneumothorax. In the more slowly progressive cases of pulmonary tuberculosis perforation of the pleura is anticipated by the formation of inflammatory adhesions—such inflammation being apparently set up by the commencing necrosis of the pleural tissue. Were it not for this, pneumothorax, instead of being a somewhat uncommon event, would be a very frequent if not an invariable incident in the course of caseous tubercle in the lungs.

In a few instances the perforation seems to take place by the extension towards the surface of a cavity itself, or by a sinus proceeding from the cavity. With very rare exceptions the disease is unilateral; and the left pleura is much more frequently the seat of the lesion than the right. Usually there is only one perforation, which may be found at almost any part of the lung. The common site, however, is the lower part of the upper lobe, or the upper part of the lower lobe; and the reason for this is that the higher parts of the lung are usually the seat of pleural adhesions, which, as we have seen, prevent perforation.

The size of the perforation varies much; in great degree according to the length of time the patient survives. It may be large enough to admit the tip of the finger, or so small as to be discerned with difficulty;

indeed, it is often not discovered at all, being overlaid with lymph which has become organised in the repair of the mischief. The opening may be direct or valvular; and these conditions have an important bearing on treatment and prognosis, as well as on the amount of suffering to which the occurrence of the lesion gives rise. When the perforation takes place, the elastic traction of the affected lung is neutralised, and the heart and mediastinum are displaced towards the sound side. If the opening be direct and free, air passes out of the pleural sac as well as into it, and there may be no intrapleural pressure; if, on the other hand, the opening be valvular, air enters the pleura during inspiration, and as the respiratory movement is reversed, the valve closes so that no air can escape: the consequence is that the pleura gradually becomes as full of air as bulging of the chest, shrinking of the lung on the affected side, depression of the diaphragm, and displacement of the mediastinum will permit. (*Vide* article "Intrapleural Tension," p. 335). The quantity of gas present depends on various circumstances—chiefly on the presence of serum or pus in the pleura, and the condition of the lungs themselves, especially of that which is perforated. Adhesions and consolidation tend to minimise the quantity, while the opposite conditions favour the largest possible accumulation.

The gas itself, as regards its chemical composition, very much resembles expired air; it consists of nitrogen with oxygen and carbonic acid, together with sulphuretted hydrogen in cases where a fœtid liquid is also present in the pleural cavity. The proportion of oxygen and carbonic acid may vary from time to time; but this matter, however interesting, is of no practical importance.

When a rupture of the pleura is due to one of the simpler causes—such as injury or the giving way of an emphysematous vesicle, the opening is soon closed, the air becomes absorbed, and the previous state is completely restored. But it is different with the tuberculous perforation: here, owing to the leakage of septic liquid from the pulmonary cavity into the pleura, acute inflammation of the pleural membrane is set up, which may be both intense and widespread. Following this comes more or less rapid effusion, which is most likely to be purulent.

On post-mortem examination the escape of pent-up air, when the cut is made through the chest wall for removal of the sternum and rib cartilages, may bear witness to the intrapleural pressure which sometimes exists. The mediastinum and heart are displaced towards the sound side; and shrinking of the affected lung, much or little according to its condition as regards intrinsic disease or adhesions, will be observed. Where the pneumothorax has lasted for some time the pleural surfaces are covered with quantities of lymph (the result of the pleurisy), which, as before mentioned, may render the discovery of the perforation difficult or even impossible. In ordinary cases the lung may be adherent in part to the chest wall at the apex, and may be the seat of cavities and of nodules of caseous tubercle. The opposite lung may show a similar state; or, if the perforation have occurred early in the history of the tuberculous condition, it may be perfectly sound. The pleura contains serous fluid or pus, the quality of the liquid as well as its quantity depending to some extent upon the time which has elapsed since the occurrence of the perforation. Exceptions to this rule, however, may be found in cases which have proved rapidly fatal, as there may not have been time for an obvious effusion to take place.

Symptoms.—In the ordinary case the patient, perhaps during a fit of coughing, is attacked by agonising pain in the chest, a feeling as of something having given way, and perhaps of fluid trickling down inside his chest, together with great difficulty of breathing. Any of these symptoms, however, may be wanting; in some cases all of them may be comparatively inconspicuous. Nor will this appear strange when we consider that the accident, as it may be called, of pneumothorax often occurs in patients already acutely ill, with rapidly caseating or softening tubercle, probably confined to bed, and suffering from respiratory discomfort and thoracic pain. Pulse and respiration rate are both increased, the latter more so than the former; the patient is cyanosed, the expression anxious, the alæ nasi working, the heart palpitating, the extremities cold, the voice weak, the temperature lowered, and the body bathed in cold sweat—in fact, as regards his general condition the patient is in a state of collapse. Dyspnœa, which is perhaps the most characteristic of the symptoms, is often extreme and distressing, the patient feeling as if he were about to be suffocated. It is most marked when the perforation through the pleura is valvular, because the condition producing dyspnœa is aggravated with every inspiration; and it may readily happen, especially if the function of the opposite lung be impaired by disease, that the case may speedily have a fatal issue. The decubitus of the patient varies a good deal in different cases. There may be orthopnœa, or he may lie half propped up on the back, or on either side. In a case recently observed the position chosen was semi-prone towards the sound side, with the head low.

Physical signs are often more definite than the symptoms. The following points are to be noted:—

Inspection.—The shoulder of the affected side is elevated, the intercostal spaces partially or wholly obliterated, the side distended, and the movements of respiration diminished or altogether absent. The respiratory movements of the sound side are correspondingly exaggerated. The heart's maximum impulse may be seen displaced towards the sound side; although, owing to the rapid and disturbed respiratory movements together with the weakness of the heart's action, it may be difficult to make out.

Palpation.—This means may enable the last-named point to be more distinctly perceived; and by it we can also appreciate the diminution or abolition of respiratory movements: tactile fremitus is also abolished. Displacement downwards of the liver or spleen may be observed according to the side affected; and the displacement may be very considerable in amount if the pleural cavity contain much air or liquid, or both. This change has an important bearing upon treatment, since downward displacement of the diaphragm forms such a large pocket for the accumulation of pus that its amount is very apt to be underestimated; thus steps for its prompt removal may not be taken.

Percussion.—The presence of air in the pleura gives rise to a marked change in the percussion resonance; the note is over-resonant, and may generally be described as tympanitic. When the tension of the walls, however, becomes very great, there is a change in the note, so that it is shorter and of higher pitch, and hence of a less tympanitic quality. The "cracked pot" sound might be expected in cases where the perforation between the lung and pleural cavity is open and free; and some writers state that it is present occasionally, although rarely. The characteristic note may not be made out over the whole of the affected side; adhesions fixing a portion of lung to the thoracic wall may prevent it, and this condition is of course most frequently observed at the apex of the lung. Or the presence of an accumulation of liquid—purulent or otherwise—at the base of the pleural cavity will cause a dulness in the percussion note over the area so occupied. In the latter case the dulness and tympanitic resonance may be made to alter their relative positions by changes in the position of the patient's chest.

The normal area of cardiac dulness is abolished in cases of left-sided pneumothorax; and in any case, owing to great displacement of the mediastinum, the tympanitic note often encroaches considerably on the sound side.

Lastly, there is what is known as the bell sound, the "bruit d'airain" of Trousseau, an interesting phenomenon which may be said to belong partly to the domain of percussion and partly to that of auscultation. It is recognised when some part of the side which is distended with air is auscultated, while a coin placed on another part is struck with another coin or some similar hard substance, such as a key. The sound conveyed to the ear of the listener is of a ringing metallic quality often closely resembling the tinkling of a small bell.

Auscultation.—When the opening is valvular, and the pleura has become as full of air as possible, no breath-sound may be audible, except perhaps along the spine where the compressed lung lies; but when the opening is patent, breathing of an amphoric quality is well heard, as a rule, both with inspiration and expiration.

It was formerly thought that there must be a passage of air through the perforation in order that breath-sounds may be heard, but this opinion is no longer held. If air enter and leave the lung at all, as it may do in parts where adhesions have prevented complete collapse, breath-sounds of the quality referred to, although distant and feebly conducted to the ear of the observer, may often be heard, even through the pneumothorax.

The amphoric breath-sound, when present, is most likely to be easily detected just over the site of perforation. Voice and cough sounds have a metallic ring in cases where the opening into the pleural cavity is free; and, in connection with the cough especially, the phenomenon known by the name of "metallic tinkling" is often well heard. It is not due, as was thought by Laennec, to drops of fluid falling in the air-filled cavity; but it may be produced by various adventitious sounds having their origin in the lung.[1]

Finally, there is the succussion sound, often associated with the name of Hippocrates, because it was first described by him. To elicit it, the patient, preferably sitting up, is sharply jolted or shaken, while the observer has his ear applied to the chest; or, if not acutely ill, he may be made to shake himself so as to bring out the sound. It is caused by the splashing of the liquid effusion in the cavity containing also air, just as it would be produced in a cask having similar contents; and it is of the metallic ringing quality which characterises all the adventitious sounds of pneumothorax. The patient himself may be conscious of the presence of fluid in his chest, while under examination he may both hear and feel the splashing of the fluid.

Diagnosis.—As many of the phenomena accompanying the majority of cases of pneumothorax are of a definite and striking character, the diagnosis, generally speaking, is not a matter of much difficulty. The essential points are: over-resonance; absence or great enfeeblement of breath-sounds (these, if present at all, being of amphoric quality); displacement of the heart, and the bell sound. These are perhaps more than enough for diagnosis; and they are necessarily strengthened if we have a history of sudden attack of pain in the chest with dyspnœa. The only class of cases at all likely to give rise to doubt are those in which the pneumothorax is partial, and limited by old adhesions between the pleural layers.

From emphysema, which in some points may seem to resemble pneumothorax, the distinction is easily made by the fact that emphysema is bilateral, and that in it there is no lateral displacement of the heart and no bell sound; also, that the resonance of emphysematous lung is not so tympanitic as is the rule in pneumothorax. It must be admitted, however, that rare instances occur in which the distinction is a fair point for discussion. I can recall two such cases: the diagnosis of emphysema, however, was duly made in both cases.

From a large pulmonary vomica pneumothorax is distinguished by the absence of the bell sound, a duller quality of resonance, even where the conditions of the cavity are most favourable for confusion of diagnosis, and the absence of displacement of the heart; or, at any rate, if the heart be displaced, it is towards the affected side, and is due to contraction of the lung. The side of chest affected would also be rather retracted than distended. Metallic tinkling and amphoric breath-sounds may, of course, both be obtained in cases of cavity; and, indeed, even the succussion sound, if the cavity be large and contain a quantity of liquid.

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[1] In a case seen by me about fifteen years ago in a healthy, athletic young man of some twenty years of age, the air escaped into the pleural cavity with a succession of tinkles or clicks. These were audible in all parts of the large room, and continued until the family medical attendant arrived, probably two hours, so that he also heard them plainly. Before my arrival they had ceased. I suggested that the sounds were due to a rupture of a tiny bubble at each issue of air. Their frequency varied, they came much faster at first and grew rarer. Inspiration, at any rate at first, increased the number and loudness of the tinkles. The rupture was brought about by an attempt to bend the body backwards so as, if possible, to touch the ground with the hands without removing the toes from a line. The patient, whose pneumothorax on our examination was considerable, soon got well and has remained well.—ED.

From pyopneumothorax subphrenicus—the name given by Leyden (10) to a condition in which an abscess cavity receiving air through a fistulous perforation from an air-containing viscus (most commonly perforating ulcer of the stomach) is found below the diaphragm, the principal guide to diagnosis is to be found in the history of the case.

Lastly, resonance and breath-sounds somewhat resembling those of pneumothorax are occasionally found at the apex of the lung in cases of pleural effusion; and sometimes over part of a lung consolidated by pneumonia. The site of the physical signs here, and a careful estimation of the condition generally, will probably prevent any mistake in such cases.

A few other rare conditions, such as hernia of a part of the stomach, or colon, through the diaphragm, have simulated pneumothorax: such an accident is usually the result of injury, and it can generally be distinguished without much trouble.

Should there be any difficulty in deciding on the causation of a case of pneumothorax, the withdrawal of a few drops of fluid from the pleural cavity, if such be present, and its examination for tubercle bacilli may be of material help. This was done in a case recently under my own care, and it furnished positive results. Careful attention to the physical signs and symptoms of the case will probably enable the physician to arrive at the correct conclusion as to the nature of the perforation in the lung, which is important from the point of view of treatment.

Prognosis.—The prospects in a case of pneumothorax depend chiefly on its cause. In the simple and traumatic class of cases the opening soon becomes sealed by inflammatory exudation and the air is absorbed. In all the other varieties prognosis must be guided practically by the underlying disease. The tuberculous cases, which, as we have seen, form a large majority, end for the most part unfavourably, and that at no distant date. The shock and intensity of the early symptoms may even cut life short in a few hours. At the same time, much depends upon the condition of the opposite lung, as well as upon the presence of adhesions limiting the extent of the pneumothorax in that which has become perforated. Although it may seem paradoxical to say so, patients who, before the occurrence of the pneumothorax, had been in comparatively sound condition are, so far as pneumothorax is concerned, in greater danger than those whose affected lung has been much crippled by disease; and this is chiefly due to the fact that in the latter case the system has gradually adapted itself so far to its changed conditions as to tolerate an amount of interference with normal function which would excite much greater disturbance if it fell upon the patient with all its force suddenly. The same thing is seen in cases of ordinary pleuritic effusion. If this occur very slowly, the physical signs may indicate that one side is practically full of fluid, and no respiratory distress, apart from exertion, may be complained of; while a second case in which half the quantity of fluid is present may be characterised by great dyspnœa if the accumulation have been rapid.

Both clinical and pathological experience go to show that even in tuberculous cases of pneumothorax rare cures have taken place; but in the great majority the outlook is a very dismal and discouraging one.

Treatment.—In most cases this can only be palliative and symptomatic. So far as drugs are concerned, opiates and stimulants comprise practically all the medicines likely to be useful. Morphine, either by the mouth or subcutaneously, is perhaps the best of the former class; alcohol in some form of the latter, but its effects may be helped by ether and ammonia. The opiate acts beneficially by relieving pain, checking the cough, and diminishing the discomfort of the patient generally, especially that resulting from the dyspnœa; and the stimulants are called for both to counteract the collapse first occurring, and to help the heart to carry on its work in which it is handicapped both by the alteration in its position and the obstruction of the circulation through the compressed lung tissue. Some external applications are useful. Dry cupping may be recommended if the dyspnœa and cyanosis be great; and where pain, resulting from the accompanying pleurisy, is much complained of, the application of two or three leeches and hot fomentations are likely to give relief. Subsequently strapping the side may be thought of.

Sooner or later the question of paracentesis will, in most cases, have to be considered. If there be evidence that the pressure within the thorax is considerable, we have practically no choice; especially if, owing to the valvular character of the perforation, this pressure be increasing. A fine trocar should be used, but no aspiration. The danger, of course, is that the diminution of the intra-thoracic pressure may encourage the reopening of the perforation which may have been closed by lymph, a condition on which our hopes for a cure of the pneumothorax depend; but it is better to run this risk than to allow the patient to die from asphyxia and exhaustion. If the opening should not have closed, the passage of the trocar will at least do no harm, and it will enable the presence or absence of intra-pleural pressure to be demonstrated. After puncture, strapping of the affected side, in order so far to prevent the recurrence of distension, may be employed in some cases with advantage. The only danger which attends puncture is that subcutaneous emphysema, partial or general, may spread from the seat of it; but this rarely happens, and all risk may be practically abolished by keeping up a little pressure on the wound after the puncturing instrument has been withdrawn.

In any case when there is evidence that the pleural cavity is partly occupied with liquid, it is wise to explore from time to time to ascertain the nature of the liquid. If serous, the general condition of the patient will be no worse than if air alone were present; probably indeed better, as the pressure exerted on the lung may tend to check the progress of disease in it, and will promote the effectual sealing up of the perforation. If the liquid be fœtid pus, nothing but harm can come from letting it remain in the pleura, and it ought to be freely evacuated at once. But there is an intermediate class of cases in which the fluid is purulent, not fœtid; and it is more difficult to decide what should be done here, and when. In such a case, if the pneumothorax have resulted from the rupture of an empyema into the lung, the chest should be freely opened and drained; and the same would hold good if the empyema had ruptured through the chest wall, the opening which nature makes not being, as a rule, sufficient for free drainage. And even in the case of pyopneumothorax of tuberculous origin, a consideration of general principles dictates the free evacuation of the pus, the case being thus converted into an empyema with some chance of the perforation in the lung being closed, followed by slight re-expansion of lung and obliteration of the pleural cavity. It is true that tuberculous patients in whom this is done rarely recover; this, however, is not because of the removal of the pus, but of the progress of the disease which produced it. On general grounds it is something of an opprobrium to allow a patient to die with a large quantity of pus in his chest.

The diet should be light and nutritious, and the bowels must not be allowed to become constipated. The treatment does not differ otherwise from that of phthisis pure and simple.

The question of *prophylaxis* is a more difficult one, and has reference, of course, almost solely to tuberculous cases. In them, as has been pointed out by Dr. Henry Thompson (14), there may be a warning of coming danger. His view is that a hint of impending perforation may be found in a persistent and prolonged decubitus on one side, on account of pain and cough when lying on the other side is attempted; and that such a condition suggests the presence of cavities underneath a part of the pleura unprotected by adhesions; for with adhesions there would be no such severe and continuous pain. Under such circumstances strapping of the side is more than ever advisable; medicines should be administered to keep down the cough, which in these cases is apt to be frequent and exhausting, as well as superfluous: this form of cough, says the author, "is imminently dangerous from the strain it puts upon the damaged lungs, and upon their frail investing membranes." Every physician must have seen cases which correspond exactly to his description.

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D. W. F.

N.B.—Dr. Frederick T. Roberts's article on "Diseases of the Mediastinum and Thymus Gland" has been carried forward to the end of the Diseases of the Chest, in the sixth volume, where fuller justice can be done to the subject.

DISEASES OF THE CIRCULATORY SYSTEM

GENERAL FEATURES OF THE BLOOD
METHODS OF CLINICAL EXAMINATION OF THE BLOOD
CARDIAC PHYSICS
CHLOROSIS
PERNICIOUS ANÆMIA
SPLENIC ANÆMIA
HÆMOPHILIA
HÆMORRHAGES IN NEW-BORN CHILDREN
PURPURA
SCURVY
  INFANTILE SCURVY
HÆMOGLOBINURIA
LEUCOCYTHÆMIA
DROPSY
HEART DISEASES—
  CONGENITAL MALFORMATION OF HEART
  DISEASES OF THE PERICARDIUM
  DISORDERS OF FUNCTION, INCLUDING STRAIN
  INJURIES BY ELECTRIC CURRENTS OF HIGH PRESSURE
  DISEASES OF THE ENDOCARDIUM
  DISEASES OF THE MYOCARDIUM
  CHRONIC VALVULAR DISEASES—
    DISEASE OF THE AORTIC AREA OF HEART
    DISEASES OF THE MITRAL VALVE

To be completed in Volume VI.

GENERAL FEATURES OF THE BLOOD

In the following pages I propose to treat in a very general way of such of the salient features of the blood as are likely to be referred to, or ought to be attended to in discussions on the nature and treatment of disease. A very large number of these features will in succession be necessarily discussed in detail by my brother contributors in connection with various diseases, and I must content myself with a very rapid survey of the whole field.

I begin with a remark which, though exceedingly commonplace, ought always to be distinctly borne in mind. Blood (and when we use the general phrase blood, we mean blood as it is discharged from the heart, not blood taken from any particular blood-vessel) is within very narrow limits uniform in composition and character under very varying circumstances; but that uniformity is the result of the delicate balancing of the many changes which the blood undergoes in nearly all the several parts of the body. As it flows through the capillaries of each of the tissues the blood puts on special characters, so that the blood of one vein differs, and may differ widely from the blood of another vein; but the changes thus brought about are of such a nature, and are so adjusted by a variety of influences, that the mingled blood of all the veins as it issues from the heart is under normal circumstances the same. Any marked alteration in the features of the blood flowing from the left side of the heart means something wrong in the tissue changes or some disturbance of compensatory influences.

Another general preliminary consideration deserves attention. The corpuscles are the only independent intrinsic constituents of the blood, the only idiohæmic elements. While the constituents of the plasma are continually passing through the capillary wall to and from the tissues, the corpuscles as a rule remain within the blood-stream. The red corpuscles, born in corners of the stream, in the red marrow or elsewhere, never leave it save under the most exceptional circumstances; the white corpuscles may at times wander out of it, but do not leave it to any great extent, at least save under special influences; whereas all the constituents of the plasma are continually coming and going. And while the circumstances determining the entrance and exit of the latter, whether these be activities of the tissues, or the physical conditions of the circulation, are very largely under the dominion of the nervous system, the behaviour of the former cannot be directly influenced by it; it is only in an indirect way that any nervous event can affect a corpuscle of the blood.

In connection with this aspect of the blood it ought to be remembered that the changes which are continually being effected in the blood have, so to speak, two objects in view. The purpose or function of the blood is undoubtedly to nourish the tissues, to carry to a tissue its appropriate food, and to sweep away from it its waste matters; and the primary object of the changes going on is to fit the blood for this purpose in respect to all the tissues. But it thus nourishes the tissues by means of the mechanism of the circulation; blood is driven in a certain way round and round the body. That it may be properly so driven certain physical qualities of the blood are necessary. For instance, the proper circulation of the blood is dependent on the blood possessing a certain viscosity; an increase or decrease of that viscosity means an interference with the due stream; and so with other qualities of the blood. These physical qualities are maintained by the action of the tissues; and this part of the work of the tissues is no less important than that part by which each tissue fits the blood for the nourishment of its brother tissues. Further, while it is true that in the act of nourishing a tissue the essential factors are the transit from the blood to the tissue of certain substances, and the transit from the tissue to the blood of certain other substances, it is no less true that the transit in each direction is dependent not only on the presence of those particular substances in the blood and in the tissue, but also on the presence of other substances which indirectly determine the transit. Thus to take as an illustration an extreme case:—granted that in any case the essential fact of the nourishment of a tissue is the transit from the blood to the tissue of sugar, that transit will not be the same if the sugar is offered in simple aqueous solution, as if it be presented as part of the compound plasma of blood. Most probably, in the internal struggle for existence, the economical result has come about that the substances which are actually employed for the nourishment of the tissues are, to a large extent, also used for maintaining these other qualities of the blood. But it is also probable that such an economy is not complete. Indeed it may be regarded as an open question whether the meaning of the large proportion of serum-albumin in the plasma is not to be sought for in the presence of this body in such quantity being necessary for the adequate flow of blood through the blood-channels, and for the proper transit of proteids other than itself and of other substances from the blood to the tissues, rather than in the nutritive value of the substance itself.

Again, we have evidence that the blood protects the tissues against the action of bacteria and like organisms. In this aspect it governs the nutrition of the tissue in a way different from either of the above.

In any case, in discussing the harm which may accrue as the result of any change introduced into the blood, we ought to bear in mind that the harm may be wrought in one or other of the ways to which we are calling attention. Likening the blood to a medicament, we may say that it may fail not only through error in the quantity or quality of the active drug, but also through the vehicle or medium being unsuitable.

Bearing these considerations in mind, we may inquire what are the several changes which may take place in the blood. And we may first turn to the changes of blood as a whole.

Volume or quantity of blood.—The space supplied by the combined vascular channels contains a quantity of blood, which under ordinary circumstances is in man calculated to be equal in weight to about one-thirteenth of the whole body. This mode of stating the quantity presupposes a certain normal composition and specific gravity, as indeed does also the method of determining it; since the quantity remaining in the body after all that can be shed has been shed, is calculated upon the amount of hæmoglobin retained in the body.

We have in fact no accurate observations on the volume of blood irrespective of its composition. Probably the mere volume is of no great moment. As a mere store of material it contains probably a surplus of everything, and a little less or a little more of the whole surplus cannot produce any great effect. Of more importance is the volume in relation to the total capacity of the blood-channels. But this capacity is variable, and by vaso-motor action can be so adapted to the quantity present, at least within certain limits, that the rate of flow and the pressure on the capillary walls remains, within limits, the same with varying quantities of blood. Hence, also, within limits, neither the addition nor the withdrawal of blood produces any marked change in the blood-supply to the several tissues. Neither plethora, in the old sense, nor its opposite, has any physiological significance. At the same time it must not be forgotten that an excess of blood may lead to an accumulation in the venous channels on the other side of the capillaries, and while not directly affecting the supply to the tissues, may produce physiological effects.

Reaction.—Since blood is the great agent of the chemical changes of the body, the chemical reaction of the blood as a whole assumes great importance. The chemical changes wrought by means of the blood must be influenced by the blood being alkaline, neutral, or acid; and by the amount of its alkalinity or acidity. Normal blood is alkaline, the alkalinity in man being equivalent to that of from 320 to 390 mgms. of sodium carbonate for every 100 grms. of blood. This normal condition is like the other normal conditions of the body, an equilibrium established between contending processes and hence complex in origin. The reaction actually tested is that of the plasma, but this is governed by processes taking place in the red corpuscles; for these bodies, by virtue of changes taking place in them, can give up to or take from the plasma substances whereby the alkalinity of the latter is increased or diminished. It is apparently through the action of the red corpuscles that the alkalinity of the plasma (or serum) decreases after blood is shed, and that an increase of carbonic anhydride in the blood actually increases instead of diminishing the alkalinity.

This complexity is illustrated by the experience that the alkalinity, while it may be diminished by the continued administration of acids, cannot be correspondingly increased by the continued administration of alkalies; that it is not influenced by the secretion of gastric juice, and that the diminution of alkalinity by violent exercise is less than that by moderate exercise, being moreover largely dependent on the proportion of proteid matter in the food taken at the same time. The alkalinity of the blood is part and parcel of the alkalinity of the tissues in general; it is not the consequence of the alkalies in the food being in excess of the acids, but comes about because the general metabolism of the body results in an alkaline reaction. The white corpuscles appear to have no share in the matter, but the red corpuscles, as we have just said, do seem to play a part.

Specific gravity.—Since the corpuscles are heavier than the plasma, the specific gravity of the whole blood may be affected by a mere change in the number of red corpuscles. It may, of course, also be affected by a change in the density of the plasma and corpuscles (for a change in the one would bring about a change in the other) without any change in the number of corpuscles. Or a change in the number of corpuscles and a change in the density of the plasma might occur at the same time, the change in the two factors being in the same or contrary direction. As a rule, perhaps, a low specific gravity is accompanied by a scantiness of hæmoglobin due, most frequently, to a diminution in the number of red corpuscles, but sometimes to the corpuscles containing less hæmoglobin.

In health the determination, by various methods, of the specific gravity of the blood has given results varying from 1050 to 1060, the more common result being about 1056. Though the blood in different veins may differ in specific gravity, the venous blood is not materially or uniformly denser than arterial blood. The blood of the pregnant female is of low, that of the fœtus or new-born animal of high specific gravity. In certain diseases, especially in anæmia, and particularly in pernicious anæmia, the specific gravity may be very low; cholera and diabetes are the only diseases in which it is known to rise beyond the limit observable in health. It is worthy of notice that any obstruction to the flow along the vessels at once distinctly increases the specific gravity. Hæmorrhage lowers it, but the normal or nearly the normal is very quickly regained. Dilution of the plasma by the injection of innocuous dilute fluids, normal saline fluid for instance, similarly lowers it; but the effect also soon passes off, and though by repeated injections a low specific gravity may be maintained for some time, yet a rise to the normal or nearly to the normal speedily follows the last injection. The return to the normal after dilution may be explained by the escape of water from the interior of the vessels, and this appears to take place chiefly in the splanchnic area. The interpretation of the effects of hæmorrhage is not so clear. We may suppose that the lost volume of blood is in the first instance replaced by plasma only. Though we know that hæmorrhage stimulates the production of new red corpuscles, it is difficult to believe that these can make their appearance in sufficient numbers to account for the regained density, seeing that this may occur within less than half an hour. It looks as if the system got rid of the water of the extra plasma, and regained its density by acquiring a smaller volume.

Whatever view we take of the nature of the process of transudation of lymph, a lowered specific gravity due to a diluted plasma must, by the changes in the osmotic powers, influence that process, and so the nutrition of the tissues. Hence the effect of a low specific gravity thus caused will be different from a low specific gravity due merely to a scantiness of red corpuscles; the latter will also have effects, but of a different kind. In attempting, therefore, to explain any feature of disease by reference to a low specific gravity, it becomes important to ascertain the exact way in which the specific gravity is lowered. Further, a change in the osmotic powers of the plasma directly affects the corpuscles; in this way actual destruction of the red corpuscles (hæmolysis) may be brought about. Again, when the specific gravity of the blood is raised by a relative increase of red corpuscles, the resulting increase of friction, both internal friction of corpuscle with corpuscle, and friction of the corpuscles against the vascular wall, affects the physics of the circulation; such a blood is driven along with greater difficulty.

We may now turn to the particular changes of the blood either going on continuously or taking place from time to time. These fall into two categories: the changes effected by the several tissues on the blood as it is passing through the capillaries, and the events which occur in the blood-stream itself.

1. *The changes effected by the tissues.*—These may be subdivided into three main groups: those effected by the advehent tissues which, through the alimentary canal,[1] bring material to the blood, by the excretory tissues which remove material from the blood, and by the metabolic tissues which change the blood as it passes through them.

To deal in detail with the normal changes so effected would be to traverse a great part of physiology, and to deal with the abnormal ones would be trespassing on the fields of others; with regard to the advehent tissues it may, however, be worth while to point out some of the more striking ways in which something wrong in their action may, by modifying the blood, work mischief in the animal economy.

Assuming that proteids are chiefly converted into peptone and albumose, but to some extent into leucin and other deproteonised bodies, that the peptone and albumose, in the act of absorption, are converted in one or other of the natural proteids of the plasma, and that all the digested proteids are carried to the liver, there to undergo a secondary digestion before they are thrown on the blood-stream, the following are some of the errors of digestion to be borne in mind as possible causes of disease, apart from mere excess or deficiency of normal action. The proteids may be absorbed in some form other than as peptone or albumose. The conversion of the peptone or albumose into the natural proteid of the plasma may fail or may take the wrong direction. The elements of the proteid converted into leucin and other substances may be in excess or may be deficient. Some one or other of the digested proteids may pass into the lymph-stream instead of into the portal blood, and be thrown into the general blood-stream in an unprepared condition; the importance of this diversion is shown by the fact that when in the dog the portal blood, instead of being allowed to pass through the liver, is carried, artificially, into the inferior vena cava, proteid food has poisonous effects.

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[1] We may in this relation neglect the entrance of oxygen through the lungs.

Our knowledge concerning the digestion of fats is at present very imperfect and uncertain. But assuming that all the fats pass into the lymph-stream and not into the portal blood, and are carried in the lymph-stream in the main as neutral fats (having been synthesised again by the epithelium, even if, in the very act of absorption, they have been split up into fatty acids), but partly as soaps, and that both are subjected to some unknown influences during the passage along the lymphatic tract, we may assume that the blood will not be the same if the fats should find their way into the portal blood, or if the proportion of neutral fats to soaps should be changed, or if the lymphatic tract should fail to exert its normal influence on the fats during their passage along it.

Assuming that carbohydrates are converted into maltose, and that this is chiefly absorbed as dextrose into the portal blood, but partly undergoes a further change, by fermentation, into lactic or even into butyric acid, the following are some of the errors to be borne in mind. The conversion of maltose into dextrose may fail or come short, or some sugar other than dextrose may be formed. The sugar formed, of whatever nature, may pass, not into the portal blood, but into the lymphatics, and be thrown on the general blood-stream without having passed the gauntlet of the liver; and this seems a possibility especially well worthy of notice. The proportion of carbohydrate converted into lactic or other acid may be in excess, or deficient; and abnormal acids may be formed and pass into the blood.

With regard to the excretory tissues, I will content myself with the remark that these fall into two classes in respect to the influence which they exert on the blood. On the one hand—as in the case of the kidney and urea—the act of secretion may be the simple one of picking up from the blood a substance already existing in it; variations in the activity of the excretory organ in such a case have no other effect than that of removing more or less of the substance from the blood. On the other hand, the act of secretion may be more complex and include metabolic activity; the substance excreted is formed in the excreting organ previous to its ejection; as, for instance, in the secretion (which, so far as the blood is concerned, is excretion) of pepsin or the bile acids. In such a case, in the act of secretion the antecedent metabolism may go wrong and the blood in consequence be affected.

To deal with the changes in the blood effected by the metabolic tissues would lead me through a great part of physiology and into discussions concerning the nature of many diseases. I must confine myself to one or two general reflections.

Certain tissues have what may be called an outward function, by which they affect parts other than themselves, such as a muscle in moving a limb, a secreting gland in pouring forth its secretion, and so on. In such cases the metabolism is the means of carrying out the function, and may be considered as brought about for that purpose. This is conspicuously the case in a muscle. Undoubtedly the muscle in contracting pours into the blood, either directly or indirectly through the lymph-stream, what we speak of as waste products. These waste products, like other waste products, may be capable of utilisation, but they are produced, not for that secondary purpose, but because they necessarily result from the act of contraction. Other tissues, of which the suprarenal capsule may be taken as an example, have no such outward function. They undoubtedly produce changes in the blood, but these changes serve no purpose in the organ itself. They are brought about for the sake of the blood itself; the blood so changed serves useful ends in the economy. Other tissues, again, afford a combination of these features. And, indeed, perhaps the distinction just made is after all not a valid one. Each of the tissues becomes adapted to thrive on the blood as affected by the other tissues. Hence, even admitting that the metabolism of muscle was in the first instance directed merely to give rise to contraction, and so to movement, the products of the metabolism being also in the first instance carried to other tissues merely to be prepared for excretion and excreted, it might easily arise that some turn in the muscular metabolism consistent with the efficacy of the muscle as an engine of movement was of advantage to one or other of these secondary tissues. If so, this would in the course of development become fixed and exaggerated. Hence we may be wrong in supposing that muscular metabolism is shaped solely and exclusively for the good of muscular contraction. And, indeed, we have hints that it is not. The hygienic effects of muscular exercise are manifold and far-reaching; it brings about changes in the circulation and respiration which have indirect effects on the other tissues quite apart from what is going on in the muscles themselves. Still it is difficult to resist the suspicion that the good effects are in part due to the actual metabolism of the muscle itself; whether it be that substances in the blood arising in other tissues are drawn into the complex vortex of that metabolism and made use of in an anabolic way, or whether some of the stages of the muscular katabolism, and we may well believe that these are many, cast off into the blood from the muscle subserve useful ends in other tissues. If there be any truth in this suggestion we may look to further study of the blood to explain, on the one hand, how disorders of the muscular system may arise from events in other tissues, and, on the other, how this or that tissue having no other connection with the muscles than through the blood, may suffer when, as in certain nervous affections, so large a proportion of the muscular system is out of order that the few which are sound cannot effect what is needed.

There is, of course, one metabolic feature which stands apart from all others in being common to all the tissues: in every tissue oxidation is always taking place, oxygen being supplied by the blood, and in every tissue carbonic anhydride is a conspicuous product of the chemical changes. This is true even of the liver, whose main blood-supply has already been used for oxidation purposes and become venous. The salient features of this respiration of the tissues, as it has been called, are two. In the first place, since carbonic anhydride is only one of the several products of the oxidative metabolism, and that metabolism complex in character, the carbonic anhydride not arising from the direct oxidation of carbon, but as the last step in a chain of events, no direct proportion obtains at any moment between the oxygen absorbed and the carbonic anhydride given out; the latter may be given out in quantity by a tissue which at the time is taking in no oxygen, and may not have taken in any for some time previously. In the second place, while the physical conditions of the lungs are such that arterial blood has always the same proportion of gases, and these sufficient to cover all the respiratory needs of all the tissues under ordinary circumstances, these needs are very variable, the several tissues differing from each other, and the same tissue having different needs at different times. Hence the respiratory activity of any tissue, the amount of oxygen it takes in, and the amount of carbonic acid it gives out, are determined by the tissue itself, not by the character of the blood. The margin of the respiratory value of arterial blood is so wide that it is only under extreme circumstances, those approaching asphyxia and such as cannot long be maintained, that the character of the blood at all affects the respiratory activity of the tissues.

2. *The changes taking place in the blood-stream.*—We may now turn to the changes which we may speak of as changes taking place in the blood-stream itself. In attempting to deal with these, however, we come upon an important preliminary consideration. In what has gone before we have had to do with the particular changes in the blood brought about by particular tissues, that is to say, by the action of the elements of the tissues lying on the other side of the wall of the vascular channel, and exerting their influences across that wall. That the tissue produces the change may be ascertained by various experiments or observations directed to this or that tissue, such as by removal of the thyroid and the like. But the blood may and indeed does undergo changes which we cannot, as yet at least, attribute to the action of any particular tissue. For instance, the introduction of a disease or toxic agent under certain conditions leads to such changes in the blood that the serum acquires in relation to the toxic agent properties spoken of as antitoxic; these properties may have a different origin in different cases, but in some cases at least are probably due to the generation in the blood of an antitoxic substance, an antitoxin. It

may be, of course, that the antitoxin is produced by the activity of some particular tissue; future researches may show this. But there are at least two other possibilities. The whole lining of the vascular channel constitutes a tissue whose influence cannot be ignored; and the corpuscles, both red and white, constitute a floating tissue of whose influence on the plasma there can be no doubt. The former may be attributed mainly to the epithelioid layer; and in the absence of any knowledge that the activities of the cells constituting this layer differ in different parts of the system,—that they are different, for instance, in the veins from what they are in the arteries, or in the large vessels from what they are in the minute vessels—we may assume that the influence is chiefly exerted in the minute vessels, where the same bulk of blood is exposed to a larger area of lining. We may probably also assume that this influence is in the active metabolic tissues more or less overridden by that of the tissue itself, and that it is most prominent in a passive tissue like the connective tissue. Such an influence cannot at present be appraised; it seems, however, obvious in the phenomena of the clotting of blood, and ought not to be ignored. In the production of antitoxins, and in respect to other changes in the blood-stream, we have to bear in mind possible actions on the one hand of the epithelioid lining, and on the other hand of the corpuscles.

We may consider first the corpuscles. These, and hence the events which they bring about, are, unlike the tissue with which we have been dealing, free from the direct influence of the nervous system. By way, as it were, of compensation, they are exquisitely sensitive to changes in the physical and chemical condition of the plasma; and the consideration of their functions largely resolves itself into a study of the manner in which they react toward such changes.

The red corpuscles.—The main function of these is, of course, to carry oxygen from the lungs to the tissues. In the lungs the hæmoglobin becomes saturated or nearly saturated with oxygen; this is given up to the several tissues according to their wants, the exact amount given up at each transit differing in different tissues and in the same tissue at different times; while the amount of oxygen in arterial blood is fairly constant, that of venous blood is very variable. By adequately increasing the partial pressure of the oxygen in inspired air, not only is the hæmoglobin completely saturated, but an additional quantity of oxygen of high respiratory value is carried by the plasma. Under ordinary conditions, however, all the oxygen used by the body is thus carried by the hæmoglobin. Hence the quantity of hæmoglobin in the blood determines the respiratory capacity, but, as we have just seen, not the respiratory activity; this is dependent on the extra-vascular elements of the tissues. This quantity is mainly dependent on the number of red corpuscles, but not wholly so. A specimen of blood having the same number of red corpuscles may contain less hæmoglobin than another specimen, the difference depending not so much on the size of the corpuscles, though these may vary somewhat, as on the amount of hæmoglobin carried by the same bulk of stroma. It has been urged that different kinds of hæmoglobin exist, one kind carrying per unit of weight more or less oxygen than another; but this is doubtful.

Though the hæmoglobin does not carry carbonic anhydride in the same distinct way that it carries oxygen, there are reasons for thinking that the former is not, as was once supposed, carried exclusively by the plasma; the hæmoglobin has some share in the matter, but the exact way in which it acts has not as yet been made clear.

The red corpuscle, however, must not be considered as simply a respiratory agent carrying oxygen and influencing the carriage of carbonic anhydride. It consists of a stroma as well as of hæmoglobin; and though that stroma has lost its nucleus, and with it the power of reproduction and other vital prerogatives, it is still alive, and is still capable of influencing the plasma. The existence of such an influence, which though it may be physical, osmotic, in its nature must depend on the condition of the stroma, is shown by the fact that the entrance and exit of oxygen are accompanied by the transit from the plasma to the stroma, or *vice versa*, of various salts, notably sodium chloride. The action of each corpuscle in this direction is of course insignificant; but the combined action of the multitude of corpuscles must not be neglected; and in tracing out the effects of diminished numbers, or other abnormal conditions of the corpuscles, regard must be had to this and other possible actions of the stroma as well as to the respiratory activity of the hæmoglobin.

Even under circumstances which are compatible with health, the number of red corpuscles in a given bulk of plasma may vary considerably. This in a great number of cases is due, not to a change in the number of corpuscles themselves, but to variations in the plasma. Nevertheless it may at times be due to the corpuscles being more or less abundant; for the mean population of red corpuscles at any one time is undoubtedly a balance between the number of old corpuscles which have disappeared and the number of new corpuscles which have appeared. Though we have no means of directly determining the average duration of life of a red corpuscle, it must be short, since the whole quantity of bilirubin secreted in the bile is supplied by the hæmoglobin of red corpuscles, and the production of this must entail a large daily destruction; and though the origin of the main urinary and other pigments is at present obscure, we ought probably to conclude that an additional destruction of red corpuscles takes place in order to provide an additional quantity of hæmoglobin for these. To meet this daily destruction a large daily birth must also be going on. We have evidence that in the adult this birth takes place in the abundant venous sinuses of the red marrow of bone, out of special nucleated corpuscles (erythroblasts) lodged there; but that it may also occur in the spleen or even elsewhere, at any rate under certain circumstances. Some observers, however, still maintain that the precursor of the red corpuscle is a minute spindle-shaped body, the hæmatoblast, not unlike a blood-platelet which, living in the blood-stream, is developed into a red corpuscle by becoming enlarged, rounded and coloured.

It is worthy of notice that the loss of blood seems to be a most potent stimulus for the activity of this process of the production of new corpuscles, whatever be its exact nature and seat. This we may interpret as signifying that the erythroblasts in the red marrow (we are here adopting the most generally received opinion) are so influenced by the changes in the plasma contingent upon a paucity of red corpuscles—so feel these changes, we may say—as to be stirred up to reproductive activity. We cannot at present explain this more fully; it seems to be one of the many instances of that response of living matter, as a manifestation of "irritability," to chemical changes in its surroundings which is denoted by the phrase "chemiotaxis."

Probably the circumstances which determine the maintenance of the balance between destruction and birth act in this chemiotactic manner; but the details of such an action and the causes of its failure in disease are at present obscure. The technique of the determination of the number of red corpuscles and the quantity of hæmoglobin will be expounded elsewhere (pp. 430 and 440), and the sources of fallacy pointed out. But we may here remark that the observations which seem to show that dwelling in a high altitude increases the number of corpuscles, and so the available stock of hæmoglobin, and thus provides a respiratory compensation for the rarefied air, do not seem to have been adequately checked in view of possible fallacies. Otherwise we might conclude that the pressure of oxygen in the plasma, as determined by the quantities of oxygen held in the red corpuscles, is an important chemiotactic stimulus for the reproductive energy spoken of above.

The details of the manner in which the destruction of red corpuscles takes place are at present obscure. There is no satisfactory evidence that the disintegration of red corpuscles which may be directly observed in the spleen pulp is the chief source of bilirubin; indeed, it seems probable that this does not at all serve as such a source, the destruction being there carried on beyond the pigment stages. Some observers maintain that the free hæmoglobin required for the bilirubin is obtained by a breaking up of the red corpuscles in the liver itself under the direct influence of the hepatic cells. But a number of facts, such as the presence of free granules in the plasma, render it extremely probable that the disintegration takes place in the blood-stream, and that the hæmoglobin and other products are strained off by the liver and other organs. We say other products, because the stroma as well as the hæmoglobin has to be got rid of; in what way this is effected and what becomes of the stroma is not at present known.

The white corpuscles.—These, though far less numerous than the red corpuscles, yet by reason of their individual activity may be regarded as exercising a more potent and a more varied influence on the general nature of the plasma, and so on the events of the body as a whole. So many facts of the life-history of the white corpuscles, such as the relation of the hæmic white corpuscles, or those of the blood proper, to the cœlomic corpuscles, or those of the lymph spaces, the circumstances attending their birth and destruction, their entrance into and exit from the blood-stream, and hence their paucity or abundance either in the general blood-stream or in particular vascular regions, have to be treated in such detail elsewhere that we may confine ourselves here to very general considerations.

Further, without discussing the relations of the various kinds of white corpuscles to each other,—whether for instance they are, so to speak, distinct species, or genetic phases of one or more forms only, assuming provisionally a distinction between the hyaline forms and the granular forms, and recognising the significance of the further division of the latter into basophil and oxyphil,—that is to say, into those which have affinities for basic and those which have affinities for acid dyes, and therefore presumably for bases and acids generally—as indicative of important differences in the metabolic processes in each, but neglecting the distinction between finely and coarsely granular as of secondary importance,—we may turn to the following considerations :—

Both kinds of corpuscles, being alive, are engaged in metabolic activities, and hence both take up from the plasma as food and give up to it as waste substances in solutions; indeed, we have direct experimental evidence of this. In this way they must be constantly exerting influences over the plasma. Besides this, those which are actively amœboid may be assumed to be occupied, as occasion demands, in taking up from the plasma particles not in solution. Again, the granular corpuscle, which seems to be the seat of special metabolic activity, such as may fairly be called secretive, may be assumed to discharge, also as occasion demands, special substances bodily into the plasma. We have direct experimental evidence of both these acts in the case of corpuscles placed in artificial conditions, for instance, in a "hanging drop," and subjected to an artificial stimulus, such as the presence of a micro-organism; and we may fairly assume that a similar behaviour obtains in the blood-stream under appropriate circumstances.

The same experimental observations show us, as indeed we might *a priori* conclude, that in the exercise of their functions these corpuscles are exquisitely sensitive to changes in their surroundings—that is, in the plasma—especially perhaps to changes of a chemical kind; so that what is called chemiotaxis plays a most important part in their life-history. By virtue of this kind of irritability they react towards changes in the plasma too minute to be ascertained by any means of physical or chemical analysis at present in our power.

Thus the white corpuscles must be considered as exerting on the plasma during their life influences the exact nature and extent of which the circumstances of the moment determine; and scanty as the white corpuscles are, these influences must be of great moment to the body, and an excess or deficiency of the white corpuscles as a whole, or of any particular kind, must affect in an important manner the qualities of the plasma, and so the welfare of the body. Relying on the experimental evidence, we may conclude that the especially amœboid hyaline corpuscles have as their work the duty of clearing the plasma of the free particles which appear in it; it must be confessed, however, that we have not adequate evidence of their performing what might be expected to be a prominent task, namely, that of clearing the plasma of the globules and spherules of fat poured into it by the lymphatics, and indeed the labours in the amœboid way of either these hyaline or other corpuscles are of less moment than that of discharging into the plasma (whether the act be considered a secretion or not) various substances destined to produce certain effects. This seems to be especially the task of the granular corpuscles; and, as we have seen, the work of the basophil corpuscles is probably different from that of the oxyphil, though we are perhaps not in a position at present to define the difference. In some cases the substances discharged into the plasma are, we have reason to think, of the nature of ferments; and thus we may see, in a general way, how a change in the plasma so subtle as to escape ordinary physical and chemical analysis may, by acting on these exquisitely sensitive organisms, give rise to the appearance in the plasma of an agent whose effects on the plasma, and so on the body, are out of all proportion to its weight or its bulk. The clotting of blood may be taken as an instance of this activity of the white corpuscles. Put briefly, the clotting of shed blood *in vitro* is due to the conversion by the agency of the body known as fibrin ferment of the substance fibrinogen present in solution in the plasma into fibrin insoluble in the plasma. The conversion is not a simple and direct one; another body or other bodies than fibrin being formed out of the fibrinogen at the same time, and the weight of fibrin formed being less than that of the fibrinogen used up. The change, moreover, is not effected at one step, a precursor of fibrin, but unlike it soluble in the plasma, being formed between it and fibrinogen. The change is further dependent on circumstances other than the mere presence of fibrinogen and ferment in a liquid medium at a suitable temperature. Thus the presence of a calcium salt is equally essential; in its absence clotting does not take place. And the presence of other substances may on the one hand hinder, and on the other hand hasten the completion of the act. As regards the nature and origin of the ferment, many observers have come to the conclusion that it is of the nature of the bodies called nucleo-proteids; and there is considerable evidence that the ferment which is absent from the blood at the moment it leaves the blood-vessels is furnished upon shedding by the white corpuscles, or by some, that is a certain kind of them, through a discharge from their bodies, which may take on the form of an explosive disintegration.

It has just been said that blood at the moment of being shed appears to contain no fibrin ferment. But the absence of clotting from blood within the lung vessels under normal circumstances cannot be due merely to the absence of fibrin ferment, since very considerable quantities of active ferment can be injected into the circulation without necessarily causing clotting. If we assume (and the assumption, though probable, is still an assumption, though the evidence that circulating blood contains fibrinogen is strong) that clotting within the blood-vessels is, like that *in vitro*, a conversion of fibrinogen by the action of fibrin ferment, we may infer from this that the blood contains, or may contain, substances or agencies antagonistic to the action of the fibrin ferment or fibrinogen. That such substances or agencies may be generated in the blood-stream is shown by the action of peptone, or rather albumose. If this substance be added to shed blood, it does not prevent clotting; injected into the circulation it does do so, not only in blood while still within the blood-vessels, but after it has been shed. We may conclude that the albumose, while circulating in the blood-stream, provokes some of the tissues so to add to or so to alter the blood as to give rise to a something antagonistic to clotting. It has been suggested that this effect is produced while the blood passes through the liver, it being asserted that the antagonistic action of the albumose is not manifested if the blood be prevented from passing through that organ. Even if we regard this particular view as not distinctly proved, the albumose effects illustrate the influence of what we may generally call "the tissues" on the process of clotting. Conversely, the presence in the blood-stream of a substance which seems to be a nucleo-albumose brings about extensive intra-vascular clotting, though the addition of it to shed blood has no such effect. The complexity of the reaction is illustrated by the fact that if the same substance be injected slowly, so that a small proportion is brought to bear on the blood at any one time, its action is reversed; it is antagonistic to clotting, and produces immunity towards its own clotting influences.

Our knowledge will not at present allow us to differentiate the several "tissues" in respect to this influence over clotting; but accumulated evidence shows that in this respect the epithelioid lining of the blood-vessels themselves plays an important part: when the inner coat, of which the epithelioid lining may be regarded as the active element, is diseased or in an abnormal condition, intra-vascular clotting takes place at the spot. The mere fact that the clotting is so limited to the spot, and does not become general, indicates of itself that the process by which the clotting is brought about is a complex one. An essential factor in the matter seems to be an aggregation of white corpuscles; and the experience that a like aggregation takes place, not only over a diseased or injured inner coat, but also over an inert body, such as a needle or thread inserted into the blood, may be taken as indicating that the corpuscles are gathered together by chemiotactic influence. By a chemical touch they recognise the difference between the normal epithelioid cell and an altered one, or an element of the connective tissue underlying the epithelioid lining, or some quite strange body. And we may perhaps also conclude that the same chemiotactic stimulus which brings them together excites them to an unwonted metabolic activity, whereby clotting comes about. But beyond this difficulties arise. The fact that the clotting is limited to the immediate neighbourhood of the exciting cause shows that the general blood-stream is not affected. We may take these phenomena as indicating that in the general blood-stream the influences antagonistic to clotting are prepotent, and that the action of each corpuscle is thus limited to its immediate neighbourhood. What that action exactly is we do not at present clearly know, and we need not discuss it here. It is sufficient for our present purposes that it illustrates the theme in hand—the possible influences which the white corpuscles may exert under the direction of their chemiotactic sensitiveness. It follows that these must not be left out of count in inquiries and discussions concerning the modifications produced in the blood-stream by various agencies; as, for instance, in the important problem how a toxin generates its antitoxin. That in the instance of clotting the effect is total and limited is probably a special feature having a teleological explanation; in the more ordinary cases, where the general welfare of the body has to be cared for, we might expect that the influences exerted by the corpuscles would be general too. And though in the same instance of clotting the corpuscles themselves do all the work, not calling in at all the aid of what we call the tissues, this does not preclude the view that in other actions the corpuscles may effect their purpose indirectly through some influences of the tissue excited by their action; whether it be the tissue lining the blood-vessels generally or the extra-vascular elements of one or another organ of the body.

The peculiar bodies known as blood-platelets are regarded by some as a third structural element of the blood; but it is still difficult to make any definite statements about these. On the whole, the evidence goes to show that they must be considered as existing in normal blood, but this in respect to our present theme is of secondary importance, since undoubtedly in abnormal circumstances they are present in large numbers. We are not, however, at the present moment in a position to state authoritatively what is their real nature; whether they are destructive formed elements, minute nucleusless corpuscles of a special kind, and therefore agents, or whether they are deposits, precipitations of a special kind, so far analogous to granules, and therefore products. In the absence of exact knowledge, it would not be profitable to attempt to inquire what may possibly be the exact nature or limits of the influences which they may exert.

Besides the changes which may be brought about by each corpuscle, white or red, in an area of plasma immediately surrounding itself, we must take into account changes induced by substances more generally diffused in the plasma, and which, since they are at least usually present in the plasma, we may speak of as proper to the plasma, and that quite irrespective of the causes which lead to their presence; whether, for instance, they are products of the activity of the tissues, having not more than a transitory stay in the blood, or whether they belong to the blood itself. For instance, the evidence is clear that the blood normally contains an amylolytic ferment, though the quantity or at least the energy of it seems to vary widely in different animals; and there is like evidence that a peptic and even a tryptic ferment are also present. Our knowledge of the more easily studied amylolytic ferment is greater than that of the others; but even in regard to this we are not as yet sure whether it is a body *sui generis*, or whether it is merely the result of a back flow from the amylolytic pancreas and salivary glands, merely passing through the blood on its way to be got rid of. The undoubted fact that sugar (dextrose) rapidly disappears even in shed blood has led some to speak of the existence in the blood of a sugar-destroying body or ferment, and the absence from or the excess of such a body in the blood has been appealed to in explanation of diabetic phenomena. The existence of such a body cannot at present be considered as definitely proved; but there can be little doubt that the plasma does contain a number of bodies, some of which may be of the nature of ferments, others mere chemical substances acting in a simple and more direct manner; and that variations in one direction or another of the quantity of such a body present in the plasma may exert a great influence on the economy, and, indeed, produce morbid symptoms. Bactericidal substances in the plasma afford an illustration of the newly discovered constituents we are now discussing.

Thus, when we have to consider the effects of introducing the blood of one animal into the blood-vessels of another, we have to take into account not only the general properties which may be regarded as common within limits to all animals, but special properties differing in different animals; and these may in large part depend on the presence, relative or absolute, of the bodies just spoken of. One practical value of the transfusion of blood seems to be to supply adequate oxygen by means of the hæmoglobin. The mere bulk of blood, as we have urged above, is, owing to the adaptive action of the vessels, of secondary importance from a mechanical point of view, unless the loss be very great; and after even great loss of blood, that which is left is probably sufficient to meet the more slowly developed needs of nutrition other than those of oxygen. Against this view may be urged the clinical experience that the injection of simple saline solution is beneficial. If this be so in cases where the loss is not too great to be compensated by vaso-constriction, the increase of the bulk by the saline must work beneficially in some other way than by restoring the mechanical conditions of the circulation. Such supply of oxygen by means of the transfused blood is of course temporary only; the foreign corpuscles soon cease to be recognised in the blood-stream; they disappear, but during their stay they have met the demands of the tissues for oxygen, until such time as an adequate supply of native corpuscles has been obtained by new formation. There are no reasons for thinking that the red corpuscles of one animal, provided that difference of size does not bring mechanical difficulties to the circulation, may not serve as oxygen-carriers to the tissues of another animal. Nor are there reasons for thinking that the substances which serve as the general basis of nutrition for the tissues of one animal—dextrose for instance, and proteids, or whatever they be—will not serve in like manner for the tissues of another animal. Differences, relative or absolute, in the salt of the plasma may render the blood of one animal unfit for another;

but probably the chief cause of the blood of one animal, for instance of a toad, being unfitted for and acting as a poison towards the tissues of another animal, for instance of a frog, is to be sought in the presence in the plasma of one or more of the bodies referred to above.

While the blood, then, is traversing any part of its circuit, making its way through the capillaries of the tissue, we may recognise on the one hand the changes which are being brought about by the tissue itself, and on the other hand those which are being wrought in the blood itself; either by means of the corpuscles, or by other bodies, by organisms or chemical substances, including ferments present in the plasma. To these we ought to add, perhaps, the influences exerted by the epithelioid lining of the blood-vessels, influences which probably are insignificant and overridden in the capillaries, but make themselves felt in the larger vessels, and may be different in different parts of the vascular system—in the veins, for instance, and in the arteries, and in different veins.

The changes effected by the tissues and those carried out by the blood itself are not, however, independent the one of the other; they react upon each other in many ways, and in all inquiries this should be clearly borne in mind. Thus, granted that the white corpuscles have as their rôle the influence which they exert on the plasma surrounding each, the very number of these corpuscles, either in the general blood-stream or in special parts of it, is influenced by changes in the body, in the tissues, or in the blood itself; and a mere change in number, even if each corpuscle maintained the same action as before, would modify the events of the body. The number of corpuscles present in the blood-stream may be altered, a hypoleucocytosis or a hyperleucocytosis may be brought about by certain treatments, and whether the alteration of the population be due to actual destruction or new growth, or to mere temporary withdrawal or flushing, the mere fact that the population is not the same must influence the events of the body; or again, supposing the population to remain the same, the action of this or that tissue may so influence the corpuscles, or a certain kind of them, as largely to modify their actions.

On the other hand, as we said above in speaking of the white corpuscles, the action of the corpuscle is not limited to its immediate neighbourhood. It, for instance, may discharge a substance or substances into the plasma, either by way of secretion, or in a more extreme case by actual disruption; and this or they may provoke this or that tissue to an altered action, and so indirectly produce a marked change in the blood.

Such effects may be especially perhaps looked for as belonging to the white corpuscles; but the theme on which we are dwelling may be illustrated by the red corpuscles. The blood of an asphyxiated animal is poisonous; that is to say, when introduced into the blood-vessels of an animal it produces effects which must be attributed, not to a mere deficiency of oxygen, but to the presence of unwonted substances in the plasma. During asphyxiation the lack of adequate oxygen so modifies the metabolism of the tissues, probably the muscular tissues in particular, that the plasma receives from those tissues abnormal products which act as poisons. This is an extreme case, the very violence of which puts a clue in our hands; but we may safely conclude that milder circumstances produce effects which, though less in degree, are on the same lines. We may infer that a deficiency in red corpuscles, or in hæmoglobin, or indeed possibly some change in the nature of the hæmoglobin, though not pronounced enough to produce direct respiratory troubles, may so influence the metabolism of the tissues that the blood becomes abnormal in other respects than its mere shortcoming as a carrier of oxygen, and so produce results in the body having apparently no connection with the oxygen-supply.

Examples like the above might easily be multiplied; but enough has been said to illustrate the important view of how manifold are the agencies, actual or latent, which work upon the blood. The apparent sameness which is the blood's salient feature is but the resultant of a multitude of actions, which in health are successfully co-ordinated to each other, but which in disease cease to fit. In attempting to track out the genesis of a malady the interweaving of these many threads of the blood's life must always be borne in mind.

M. Foster.

THE CLINICAL EXAMINATION OF THE BLOOD

Although hardly appreciated at their true importance, routine examinations of the blood are capable of affording valuable assistance in the diagnosis, prognosis, and treatment of certain pathological conditions. In a certain number of diseases, among which may be mentioned leukæmia, pernicious anæmia and chlorosis, the diagnosis may be rapidly and accurately arrived at by examination of the blood alone; while, conversely, should the appearances now known to be characteristic of one or other of these diseases not be found, they can as certainly be excluded. Many other diseased conditions there are also in which, although we may not be able to evolve a diagnosis from examination of the blood alone, yet we can often obtain evidence of much value when considered in relation to the results of other diagnostic methods.

Such examinations also throw light on the progress of disease, and furnish a means of judging the results of treatment more accurately than would otherwise be possible.

Within the last few years much attention has been devoted to the simplification of the various methods employed in the examination of the blood; and concurrently, by the introduction of more perfect instruments, a notable advance has been made in the accuracy and precision of our results. Of no less importance is it that at the present time we can carry out all the necessary details of an investigation without the expenditure of more than a few drops of blood.

While, therefore, in order to justify its title, the present article will deal only with the technique of such methods as require for their performance a minimal quantity of blood, the clinical observer may rest assured that, with due care, the ease and rapidity with which the various examinations can be carried out need involve no sacrifice of accuracy.

In systematic investigation of the blood the necessary procedure will consist of several distinct processes, as follows:—

I. Microscopic examination of—(*a*) the fresh blood; (*b*) dried and stained blood-films.
II. Estimation of the specific gravity of the blood.
III. Estimation of the colouring matter.
IV. Enumeration of the corpuscles (red and white).
V. Estimation of the alkalinity.
VI. Determination of the coagulation time.
VII. Spectroscopic examination.
VIII. Bacteriological examination by means of stained specimens and of cultivations.

As a general rule, however, it will hardly be found necessary to carry out this scheme in its entirety; although experience alone can decide to which points in any given case it is desirable that special attention should be directed.

I. MICROSCOPIC EXAMINATION OF THE BLOOD.—**To obtain blood for examination.**—The skin of the part selected, which may be either the finger-tip or the lobe of the ear, is first washed with soap and water, and then dried carefully with a clean cloth. Ordinarily it is not necessary to employ antiseptic solutions. The skin is punctured with a quick stab either of a lancet provided with a means of regulating the extent of protrusion of the blade, which has been specially devised for the purpose, or a bayonet-pointed surgical needle of triangular section, which is perhaps more likely to be at hand. An ordinary sewing-needle should be employed only in default of anything better, as, however sharp it may be, it is more painful to the patient in its passage through the skin, especially if it be pressed slowly in instead of being plunged with one quick motion to the required depth. To avoid possibility of ill effect the instrument should be sterilised in the flame of a spirit-lamp. This precaution is indispensable when a bacteriological examination of the blood is to be made. On no account must any pressure be employed to expedite the flow of blood; nor before pricking should a tape or string be wound round the end of the finger. Professor Sherrington and myself have shown that under such circumstances as these temporary stasis of the blood-flow through the capillaries, with the addition of the lymph derived from the surrounding tissues, are sufficient to bring about so rapid and profound an alteration in the composition of the blood—affecting as it does the number of the corpuscles, the hæmoglobin power, and the specific gravity—as to render any conclusion based on examination of the blood thus obtained quite valueless under such circumstances. It is true that without compression of the part it is not infrequently a matter of difficulty, especially in patients suffering from certain kinds of anæmia, to obtain more than a drop or two of blood; although this difficulty may sometimes be overcome by making the puncture a little deeper. It is in such cases especially that Cabot strongly recommends puncture of the lobe of the ear, as in cases of pernicious anæmia, in which attempts to get blood from the finger had failed, he found no difficulty in getting it from the ear. He is, moreover, of opinion that the ear is decidedly less sensitive than the finger; and that there is an advantage, particularly in the case of children, in a process which the patient cannot watch. Again, in a sleeping patient the ear is usually more accessible than the finger.

A word of caution is necessary lest the patient be the subject of hæmophilia, in which case hæmorrhage, even from the tiniest wound, is apt to be profuse and difficult to stop. Hence it is always a wise precaution to make inquiry concerning former bleedings. If, on puncturing the skin, the blood-flow is fairly free, it is as well to reject the first few drops, wiping them off with a clean cloth as they exude, so that any extraneous bodies about the seat of puncture may be washed away. All blood examinations should be made at about the same hour, in order that the results obtained may be comparable one with another; and the time of examination and the hours of the meals should be recorded. This is specially desirable when enumeration of the leucocytes is in question, as allowance can then be made for digestion. But, if possible, examinations should be carried out before the first meal of the day is taken, for thus only can any approach to scientific accuracy be obtained.

Histological examination of the blood.—It will generally be advisable to examine the blood both in the fresh state and also by the staining of dried cover-glass films, which have previously been fixed in one way or another.

Examination of fresh blood.—The first point is to ensure the most perfect cleanliness of all slides and cover-glasses. Each observer prefers his own particular method, the exact details of which may be immaterial so long as thorough cleansing is secured. The use of soap and water may be sufficient for this purpose; but it is usually desirable either to boil the glasses in a strong solution of sodium carbonate, or to wash them in a mixture of potassium chromate and sulphuric acid. In either case they must receive a final wash in alcohol. To obtain a specimen of blood, a cover-glass, properly cleansed in one way or another, is held in a pair of forceps or edgewise between the thumb and first finger, and its under surface brought down into contact with a drop of blood as it oozes from the puncture. The cover-glass is then, as rapidly as possible, laid on a glass slide, when the weight of the cover-glass causes the blood to spread out under it in a film of fairly even depth. If the subsequent examination is likely to take some time, it is well to prevent evaporation by painting round the edge of the cover-glass with vaseline or castor oil. If, moreover, the slide and cover-glass be slightly warmed before use, or if some form of warm stage be employed, alteration in the appearance of the blood will take place less rapidly than would otherwise be the case. The specimen is now to be examined under the microscope, first with a comparatively low power, and afterwards, if thought desirable, with a $\frac{1}{12}$ oil immersion lens. Microscopical examination of a specimen of fresh blood discloses the size and shape of the red corpuscles and the fashion of rouleaux formation. Thus also the relative proportions of erythrocytes to leucocytes, and the number of blood-platelets present, can be roughly estimated, and, by the appearance of fibrin filaments, the rate of coagulation. In such a preparation the filaria sanguinis hominis, and likewise the malarial parasite, may be studied in the living state.

Examination of fixed blood.—For study of the finer structure of the blood corpuscles, and more particularly of the leucocytes, it is necessary to stain the blood-film which has previously been dried and fixed. In order to obtain satisfactory results special attention must be directed to the preparation of the blood-films, so as to obtain a perfectly thin and even layer of blood, the process being carried out as rapidly as possible so as to prevent the occurrence of histological changes.

Preparation of the films.—After thorough cleansing of the cover-glasses, the under surface of one of these is brought into contact with the drop of blood as it emerges from the puncture. A minimal quantity of blood having thus been taken up, the cover-glass is gently dropped upon another; after which, with the aid of fingers, or, better still, of forceps, the two cover-glasses are again separated by a lateral sliding motion of one on the other. In this way a thin and, if the process have been skilfully carried out, an even layer of blood is left on one surface of each cover-glass. These must now be left exposed to the air until the blood-films are thoroughly dry. For this purpose it is convenient to place them, face upwards, on a slip of paper, and to cover them with a watch-glass, or with one-half of a Petri dish, so as to prevent deposition of dust on the film surface during the process of drying.

Fixation of the films.—Before staining the blood-films they must be "fixed" in one way or another; otherwise the staining solutions are apt to dissolve out the hæmoglobin from the red corpuscles, or even to wash the thin layer of blood from the surface of the cover-glass. The method of fixation advocated by Ehrlich is a long and tedious one, involving the heating of the films for an hour or more on a brass plate to one end of which a Bunsen flame is applied. The point at which the cover-glasses should be placed on the plate is estimated by noting within what distance drops of water, let fall on its surface from a pipette, rapidly evaporate instead of assuming the spheroidal state. Or, on the other hand, the cover-glasses may be placed in a hot-air oven, the temperature of which is maintained at about 120° C. for a similar length of time.

Equally good results, however, are to be obtained, according to Hardy and Kanthack, by the far simpler and more rapid method of passing the cover-glasses three or four times through the upper portion of a Bunsen flame, as is now ordinarily done in the manipulation of cover-glass specimens in bacteriological work. Other observers, again, fix blood-films by methods other than heating. Nikeforoff, for instance, whose method is recommended by Sherrington, advises the immersion of the specimens in a mixture of equal parts of absolute alcohol and ether for periods of from five to twenty minutes. For special purposes, such, for instance, as the demonstration of karyomitosis, fixing solutions containing bichloride of mercury, picric acid, or other reagents may be employed.

To stain the blood-films.—For the method usually employed in the investigation of the histology of the various formed elements in the blood we are indebted to Ehrlich and his pupils. He not only demonstrated, in the first place, that the protoplasm of certain leucocytes contains discrete granules, but he further determined the existence of a definite relationship between the chemical constitution and the staining capacity of these cells. Thus his method and his classification of the blood-cells are based on a scheme of the micro-chemical reactions of their granules.

According to Ehrlich, the various stains employed in histological work may be divided into two main groups: (*a*) acid stains; (*b*) basic stains,—admixtures of these in certain proportions furnishing what he has called neutral stains. The stains included in the first class are classified under the term "acid," for the reason that although chemically they are neutral salts yet the staining principle is the acid radicle; in other words, the stain reacts tinctorially as a free acid.

Thus ammonium picrate is an acid dye because the picric acid is obviously the staining element, the ammonium base being inert in this respect. Of the other "acid" dyes the most useful for histological purposes are eosin, aurantia, induline, and orange G.; of these the first three stain well in concentrated glycerine solutions, while the last is generally employed dissolved in water.

Again, the "basic" stains are so called for the reason that in their case it is the base and not the acid on which the action as a dye depends.

Of the basic stains one of the best known perhaps is fuchsine, which chemically is hydrochlorate of rosaniline. Here the rosaniline is the staining principle and not the hydrochloric acid. Another most useful basic stain is methylene blue, of which the alkaline preparation devised by Löffler is of special value. Other basic stains in frequent use are methyl-violet, methyl-green, and safranin. Both "acid" and "basic" stains are employed in histological work on the blood for the reason that certain of the cell granules react to acid stains only, and are therefore called "oxyphil granules"; while others, which are more readily tinged with basic dyes, are described as "basophil." The term neutrophil is now very generally abandoned, as Hardy and Kanthack and other observers have shown that the so-called "neutral" mixtures of Ehrlich react tinctorially as acid dyes, and that the fine granules contained in the cell substance of what he has described as a "neutrophil" leucocyte are really oxyphil in their affinities. Ehrlich's original staining method has been found somewhat cumbersome and inconvenient in use, mainly on account of the length of time consumed in carrying out the various details of it. Fortunately, however, standard oxyphil and basophil reactions may be obtained by the simple and rapid modification of Ehrlich's method introduced by Hardy and Kanthack. They proceed as follows:—Thin films of blood or lymph dried in the air and afterwards passed three times through a Bunsen flame are floated on a solution of 0·5 grm. eosin in 100 cub. cent. of 70 per cent alcohol for half a minute or less (acid stain). Excess of eosin is removed by washing in distilled water. The film is then dried by gentle pressure between sheets of blotting-paper, again passed three or four times through the flame, and counterstained in Löffler's methylene blue solution (basic stain). After being again washed and dried, the films may be mounted in Canada balsam.

A description of the histological appearances brought out by the employment of this method and the system of classification based upon it will be found under the heading "Leucocytes."

Stain reactions of the blood in diabetes.—Bremer devised a modification of Ehrlich's method as an aid in the diagnosis of diabetes. He found that the red corpuscles of diabetic blood could not be stained with eosin as in normal blood; although, nevertheless, they reacted to the various so-called acid dyes. He therefore employed a special eosin-methylene blue stain, by the use of which the red corpuscles of normal blood are stained violet, of diabetic blood a greenish colour.

Lépine, however, has shown that this reaction is not absolutely characteristic of diabetes, since he has obtained it also with leukæmic blood. Bremer, partly for this reason and partly because his original eosin-methylene blue stain was difficult to prepare, has, more recently, altered and simplified the method considerably. The blood-film, after drying in the air, is exposed to a temperature of about 135° C. for from six to ten minutes; accuracy at this stage of the procedure is of special importance, as unless the temperature exceed 129° C. the test becomes untrustworthy. The slides, together with control slides made from normal blood, are next stained for about a couple of minutes in a 1 per cent watery solution of Congo red, or in Biebrich scarlet, or with the ordinary Ehrlich-Biondi stain. The specimens are then rapidly washed and dried.

With Congo red diabetic blood is usually not stained at all, while non-diabetic blood is coloured red; with Biebrich's scarlet an opposite effect is obtained, the diabetic corpuscles are stained, the normal ones are not.

Whether these different reactions are due to the presence of sugar in the blood, to an alteration in alkalinity, or to some other cause, is as yet undetermined.

The red corpuscles or erythrocytes.—Under normal circumstances the red corpuscles have the form of biconcave discs of a fairly uniform diameter of about $\frac{1}{3200}$th of an inch. When seen under the microscope in a single layer they are of a yellowish red colour and are non-nucleated. In a film of moderate thickness, shortly after removal of the blood, the corpuscles exhibit the physical peculiarity of running into small aggregations or *rouleaux*, so called from the supposed resemblance to a heap of coins.

In many forms of disease, particularly in wasting diseases, and in pernicious anæmia more especially, not only are the form and consistence of the red corpuscles liable to considerable alteration, but a marked diminution in their numbers is usually obvious on examination.

The number of red corpuscles normally present in the blood has been estimated by Vierordt at 5,000,000 to the cubic millimetre in man, and about 10 per cent less in woman. In the condition known as *oligocythæmia*, however, the number of corpuscles in the blood may become greatly decreased, sinking as low as one million per cubic millimetre; or, in severe cases of pernicious anæmia, even to half a million and under. The number of corpuscles is also diminished in leukæmia, and to a less degree usually in chlorosis and such cachexies as phthisis and the various forms of malignant disease. The number falls also, temporarily, after severe hæmorrhages, whether due to traumatism or to disease, as, for instance, after the intestinal bleeding of enteric fever. Should oligocythæmia be present in high degree, this will usually be apparent in the abnormally pale colour of the blood, and the obviously lessened number of corpuscles in fresh microscopic preparations. When the diminution in the number of corpuscles is less, the employment of more accurate methods will be necessary; and in any case trustworthy information as to the actual condition present can only be obtained by enumeration of the corpuscles by means of special instruments devised for this purpose. Under certain circumstances the *size* of the corpuscles may undergo considerable change, the diameter sometimes becoming increased to as much as double the normal length (from 7·5 μ to 10 μ or even 15 μ). This condition, named *macrocythæmia*, is apt to occur to a certain extent in any case of severe anæmia, but more especially in that form known as "pernicious." It must be carefully distinguished from the swelling of the red corpuscles which is prone to occur in hydræmic blood.

By *microcythæmia*, on the other hand, is understood a diminution in the diameter of the red corpuscles. These smaller bodies, or microcytes as they are called, are often globular in form, and of a more intense colour than normal. They are commonly present in pernicious anæmia, and occur in most other forms of anæmia, especially when severe; also in certain toxic conditions and infectious diseases, and after extensive burns and large hæmorrhages. At present, however, but little is known of their significance, and consequently no information of importance in diagnosis is to be derived from their discovery. Some observers are of opinion that they occur as the result of degeneration of the normal red corpuscles. Gram and Graber, indeed, go so far as to regard these microcytes as the result of changes in the blood after death.

Occasionally the red corpuscles undergo marked variations in *shape*, becoming pyriform, spindle-shaped, reniform, cup-like, or knobbed. A certain proportion of them, however, retain their normal form. This condition, named *poikilocytosis*—which is believed to be due in some measure to a lessened consistence in the stroma of the corpuscles—has been noticed in leukæmia and in anæmic states, more particularly in pernicious anæmia. Such remarkable variations in the shape of the corpuscles have indeed been regarded by some authors as pathognomonic of the latter disease. As is well known, crenation of the red corpuscles, giving rise to the so-called *mulberry* and *thorn-apple* forms, may occur as the result of evaporation in normal blood at a varying interval after its withdrawal from the circulation; but a little experience will prevent any likelihood of confusion with the change under consideration. *Nucleated red corpuscles* are found not infrequently in the blood in pernicious anæmia, and in leukæmia of the myelogenic kind, and again after extensive hæmorrhages. They may vary considerably in size, the presence of the larger variety (megaloblasts) being considered to be of especially grave significance.

The white corpuscles or leucocytes.—Leucocytosis.—Comparatively little was known of the differential characteristics and properties of the white corpuscles of the blood, or leucocytes as they are now more generally named, until the introduction by Ehrlich of special staining methods, by means of which the various forms of leucocytes can readily be distinguished one from another. The work of Hardy and Kanthack, and also of Professor Sherrington, in this country has added largely to our knowledge of the subject; and these observers, by simplifying the methods originally devised by Ehrlich, have rendered them more readily available for clinical research.

At the present day the name "leucocyte" has a somewhat wider significance than that of a mere synonym for the different forms of the white corpuscles which, under normal circumstances, can be demonstrated in the blood. The name as now employed includes also wandering cells, which may be found in the lymph-stream, in the serous cavities, and in the intercellular interstices of the tissues. Consequently leucocytes may be classified as—(*a*) tissue, and (*β*) hæmic.

The cells belonging to each of these classes are subdivided, again, according to their reaction to certain staining reagents, thus:—

- (*a*) Oxyphil
  - coarsely granular.
  - finely granular.
- (*b*) Basophil
  - coarsely granular.
  - finely granular.
- (*c*) Hyaline
  - large.
  - small.

The classification of leucocytes as tissue or hæmic leucocytes respectively, although convenient, is by no means definite. Dr. Gulland states that when the blood is first formed in the embryo, for a time it contains no leucocytes whatever, so that none of the leucocytes are aborigines of the blood; the coarsely granular basophil cell is found in the tissues alone, while the finely granular oxyphil cell is rarely met with elsewhere than in the blood: but, on the other hand, certain cells, which at one period of their life-history must be looked upon as tissue leucocytes, may later be discharged into the blood-stream.

Of the hæmic leucocytes *the finely granular oxyphil* variety is by far the most common normally, since it constitutes about 75 per cent of all the leucocytes present in the blood. The cell has an average diameter of 10 μ, and is vigorously amœboid. Its name is derived from the fact that the cell substance contains numbers of fine granules, which refract light to a slightly greater degree than the ground substance in which they lie. This cell can also be distinguished from other leucocytes by the irregular and multipartite nucleus, which usually appears to consist of a number of separate lobes linked together by fine chromatin threads. After death the various nuclear segments take on a more regular distribution in the cell-protoplasm, forming rosette-like masses; but, when living, the shape of the nucleus is constantly undergoing variation, for which reason it is generally described as "polymorphic." Opinions differ as to the cause of this diversity of shape of the nucleus, but, as has been shown by Professor Sherrington and others, it is most probably to be attributed to distortions produced by the extreme amœboid activity of the cell body. This cell is markedly phagocytic; but, as Professor Halliburton and Dr. Brodie have shown, it is readily killed and broken up by contact with solutions of certain nucleo-albumins. Vacuoles, probably containing fluid, are often to be seen in the protoplasm of the cell.

The finely granular oxyphil leucocyte corresponds to that named "neutrophil" by Ehrlich and his pupils; Hardy and Kanthack having shown that the former name is the more correct, since the granules, especially under certain conditions, obviously react to acid dyes. This leucocyte appears capable of undergoing multiplication in the blood-stream; but it is somewhat doubtful whether such multiplication takes place more commonly by karyomitosis, or by direct division of the cell.

The *coarsely granular oxyphil cell* differs from the finely granular variety, not only, as its name implies, in the larger size of the contained granules, but also in the larger size of the cell itself; the average diameter being about 12 μ. When examined on the warm stage it is found to be amœboid, but it contains no vacuoles, and is never phagocytic. The horse-shoe-like or reniform nucleus is fairly regular in shape. The granules contained in the cell substance are comparatively few in number and of large size. This is specially noticeable in certain of the lower animals. The granules are highly refractive, and have a marked affinity for "acid" dyes, by means of which they can be readily stained. As they colour deeply when treated with osmic acid, the granules might be regarded as fatty in nature, but they are not soluble in alcohol or ether.

The coarsely granular leucocyte has a fairly wide distribution in the various fluids and tissues of the body; but in the blood itself it does not usually constitute more than about 2 per cent of all the leucocytes present. It is, however, of special interest since, although it is not phagocytic, it apparently has certain functions of a secretory nature. Thus Hardy and

Kanthack have shown that the injection of a culture of anthrax into the blood of an animal causes a rapid disappearance of the granules in the cell protoplasm, and that such disappearance seems to be accompanied by and related to the secretion of a substance possessed of germicidal properties. Another special point of interest is that the granules of the coarsely granular leucocyte contain appreciable amounts of iron and phosphorus. It is not improbable, therefore, that, although themselves quite colourless, they are related to the hæmoglobin of the red corpuscles, which latter they also resemble in their proteid nature, marked refraction, and strong affinity for "acid" dyes. In this connection, also, it is of interest to note that the coarsely granular cell is to be found in specially large numbers in bone-marrow.

The *basophil leucocyte*, both of the finely granular and the coarsely granular variety, requires but brief mention, as under normal conditions the first form is rarely, the second never found in the blood. The finely granular leucocyte is occasionally met with for an undetermined period after the beginning of digestion, and apparently under certain pathological conditions, for Grünbaum has found it in excess in the blood of uræmic patients. This cell is of small size, spherical, and in its cell protoplasm contains numerous minute granules, which are deeply stained by methylene blue. The nucleus is irregular in shape. A sub-variety of the coarsely granular leucocyte—the "*mast-zellen*" of Ehrlich—in which the cell body is filled with large granules, which are stained of an intense violet-purple by methylene blue, has been found by Professor Sherrington in the blood of patients dying in the reaction stage of Asiatic cholera; and by myself in some cases of leukæmia.

The small hyaline leucocyte, or lymphocyte as it is frequently called, from the fact of its presence in large numbers in lymphoid tissue in all parts of the body, is about the size of a red corpuscle of the blood. It consists of a minimal quantity of protoplasm free from obvious granulation, in which is embedded a large spherical nucleus readily stained by methylene blue or other dyes.

The lymphocyte, numbers of which are continuously being shed into the blood by the thoracic duct, is apparently an immature form of cell. It is not amœboid. The number in the blood undergoes phasic variation, reaching its highest point between two and three hours after digestion has begun. The average number in the blood ranges between 10 and 20 per cent of all hæmic lymphocytes. This proportion, however, may be greatly exceeded in the form of disease known as "lymphatic leukæmia," in which the lymphatic glands undergo enlargement.

The large hyaline leucocyte, or myelocyte as it is also named, possesses a larger amount of protoplasm than the smaller variety. The nucleus is usually spherical or reniform, and fairly regular in outline. As it possesses a comparatively small amount of chromatin, it does not stain well with aniline dyes. The cell has not been proved to be amœboid, but nevertheless seems capable of acting as a phagocyte. In the blood it is less numerous than the lymphocyte, forming usually less than 10 per cent of the hæmic leucocytes. Increase in the number of lymphocytes is usually accompanied by increase in the total number of myelocytes also; this being specially noticeable in the leucocytosis which accompanies the anæmia of pregnancy, and that which ensues on typhoid fever. In lymphatic leukæmia, however, enormously as the number of lymphocytes in the blood may be increased, no such increase in that of the myelocytes has been observed. During life the cell protoplasm is apparently homogeneous, but, when dead, staining by means of methylene blue shows it to be full of exceedingly fine granules embedded in a matrix which does not take up the stain.

LEUCOCYTOSIS may be most simply defined as a condition in which the number of leucocytes in the peripheral circulation is above the normal standard.[1] It is practically impossible, however, to lay down any exact rule concerning the numbers of the white corpuscles or the excess which should be held to constitute leucocytosis. In the same normal individual variations occur at different times, and different individuals exhibit considerable range in the numbers of leucocytes in a similar volume of blood, according in some measure to the physical development and habit of life of each of them. Taking these factors into consideration, we shall not err to any great extent if we look upon the normal range in the adult as extending between a minimum of 6000 and a maximum of 10,000 leucocytes in a cubic millimetre of blood. Any number of leucocytes below the arbitrary limit of 6000 will constitute a hypoleucocytosis, or leucopenia as the condition is also named; while an excess above 10,000 would constitute leucocytosis. The ratio of white corpuscles to red is of itself of comparatively little importance, since conditions which cause an increase, for instance, in the number of white corpuscles may bring about concurrently an increase in the red.

It is, therefore, the absolute number of leucocytes in a cubic millimetre of blood which must be determined in each case. It is of no less importance, however, to determine the relative numbers of each kind of leucocyte present, as by such differential enumeration it becomes possible to discriminate the particular form of leucocytosis with which we have to deal in any given instance.

Leucocytosis may be either physiological or pathological. These two kinds may be distinguished by the fact that in the former the increase affects all varieties of leucocytes, with the exception of the coarsely granular oxyphil cell; while in the latter it is, for the most part, the finely granular oxyphil cell which is present in excessive numbers.

Physiological leucocytosis has been found in newly-born infants, in the later stages of pregnancy, more particularly in primiparæ, and during the process of digestion. Massage, as Dr. Weir Mitchell has shown, is found in many instances to induce moderate leucocytosis, and so likewise does the temporary application of cold in the form of bath.

Leucocytosis in the new-born is probably to be explained by inspissation of the blood in conjunction with more or less continuous digestion leucocytosis. Rieder and other observers have shown that hyaline cells, particularly lymphocytes, are more abundant in the blood of the new-born than are the other varieties of leucocytes. There is also a definite increase in the number of coarsely granular cells. The actual number of white cells present is considerably greater than in the adult, ranging as it does from 15,000 to 30,000 per cubic millimetre. The number rapidly declines, however, during the first two years of life, until at the end of such period the adult normal has probably been reached.

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[1] Leukæmia, or leucocythæmia, which would be included under this definition, is treated of elsewhere. See Dr. Muir's article later in this volume.

The leucocytosis of pregnancy has no clinical significance; and, since it is not present in the early stages, it is also valueless from the diagnostic point of view. Again, it is by no means of invariable occurrence, especially in multiparæ; while it is liable to occur also in cases of spurious pregnancy.

Digestion leucocytosis usually occurs in healthy persons, and comes on at a somewhat variable period after ingestion of food; beginning generally about one hour after food, increasing in amount for the next two or three hours, and afterwards gradually disappearing again in three or four hours more. In a few instances the blood of persons apparently healthy shows little or no digestion leucocytosis; this, in some cases at any rate, seems to be dependent upon chronic constipation, as Von Limbeck has shown. The effect of proteid food is especially remarkable in bringing about digestion leucocytosis; a mixed dietary is followed by a less striking result, while a vegetarian diet apparently exerts no appreciable influence. The increase in the number of leucocytes following the ingestion of a meal rich in proteids may amount to as much as 30 per cent, or even slightly more. It should be remembered, however, that the actual number present will depend on the standard normal to the particular individual during the passive phase of his digestive functions. It is always advisable, therefore, to make a control reckoning of the number of leucocytes in the early morning before the patient has broken his fast.

Starvation in man, especially when of considerable duration, has been found by Luciani and Von Limbeck to result in a notable diminution in the total number of leucocytes. Prof. Sherrington, who has studied this subject in the lower animals, finds that while this is so, yet at the same time, contrary to what obtains during the opposite phase of digestion leucocytosis, abstention from food leads to a decided increase in the numbers of the coarsely granular cell.

Diseases, especially those of malignant nature, which lead to difficulty either in ingestion or digestion of food—among which may be mentioned carcinoma of œsophagus or stomach—tend eventually to bring about not only disappearance of digestion leucocytosis, but an actual leucopenia.

Pathological leucocytoses.—For descriptive purposes these may be classified under the following heads:—(i.) Inflammatory leucocytosis; (ii.) Toxic leucocytosis; (iii.) Leucocytosis of certain infectious diseases; (iv.) Leucocytosis of cachexia and malignant disease; (v.) Post-hæmorrhagic leucocytosis.

(i.) *Inflammatory leucocytosis*, resulting from simple traumatism without bacterial infection, has recently been the subject of careful investigation by Prof. Sherrington. In his observations he was able to distinguish three definite stages of the process :—(*a*) A leucopenic phase, or hypoleucocytosis, during which the total number of hæmic leucocytes falls ; (*b*) A stage of hyperleucocytosis ; (*c*) A second but inconstant stage of leucopenia.

The preliminary stage of hypoleucocytosis affects all the different kinds of leucocytes, but perhaps the finely granular variety more especially. This diminution holds not only for a unit volume of blood, but also relatively for the number of chromocytes, and this in spite of coincident apoplasmia or inspissation of the blood.

In the second stage, that of reaction, a hyperleucocytosis occurs in which both the finely granular and hyaline leucocytes are concerned. Although this is the case there is a continued fall in the numbers of the coarsely granular variety. Finally, after a variable interval, the leucocytosis passes away, and may be succeeded by a fall which, on occasion, may again reduce the number of leucocytes below the normal average.

(ii.) *Toxic leucocytosis.*—Under this heading may be considered the results of subcutaneous, intravenous or intraperitoneal injection of various toxic substances, whether employed therapeutically or not. It has long been known that extracts, protein or dead cultures of bacteria, filtered yeast-cultures, organic substances such as fibrin ferment, hemi-albumose, peptones, nuclein, and leech extract, as also curare, tuberculin, pyocyanin, uric acid, and urates, have the effect, on injection, of bringing about a marked and rapid diminution in the number of the leucocytes. This stage of hypoleucocytosis, leucopenia, or leucolysis, as it was named by Löwit in accordance with the hypothesis advanced by him that leucocytes undergo actual destruction, is in turn followed by a hyperleucocytosis of greater or less degree, provided that the dose of toxic material received into the system be not sufficient to cause death. That the preliminary leucopenia is not due to a leucolysis has been proved by Goldscheider and Jacob, who have demonstrated that the leucocytes vanish from the peripheral circulation in consequence of their having become stored in the capillaries of the lungs. This process probably occurs in the liver and spleen also. During the second stage these leucocytes find their way once more into the general circulation, together, as certain observers believe, with others of more recent origin.

(iii.) *Leucocytosis of certain infections.*—In many of the acute infectious disorders leucocytosis has been found, more particularly in small-pox, scarlet fever, diphtheria, pneumonia, acute rheumatism, anthrax, erysipelas ; and perhaps in measles. Leucocytosis has also been described in typhoid fever ; but most observers are now of the opinion that it does not occur in the absence of complications. Leucocytosis is not apparent in tuberculosis or in influenza. The same has been said of malaria, but Dr. Billings has put on record a series of observations which tend to show that definite stages of leucopenia and leucocytosis undoubtedly present themselves in the course of this disease; although these variations, being of slight extent, have been overlooked by other observers. In pneumonia, on the other hand, the process is generally so well marked as to afford most valuable aid in the diagnosis and prognosis of the disease.

The increase in the number of leucocytes is due to increase of the finely granular oxyphil variety; it begins with the rise of the temperature, and, except in cases of extreme gravity, not only continues but increases up to the crisis, at which stage a well-marked leucocytosis is a very favourable sign. On the other hand, if leucocytosis be absent or ill marked, the case will probably end in death. In scarlet fever and other diseases, in the course of which leucocytosis is ordinarily demonstrable, we can in like manner judge in great measure of the severity and the probable termination of any given case by the extent of the leucocytosis. This being so, it is obviously desirable that the blood should be more generally examined in infectious disease than has hitherto been customary. The importance of these observations is confirmed by the work of Everard, Demoor, and Massart, who state, as the result of the inoculation of guinea-pigs with varying doses of pathogenetic organisms, that while the primary result is invariably a fall in the number of leucocytes in the blood, this in turn is succeeded by a leucocytosis in those instances in which the animal eventually recovers. They add that, in immunised animals, a leucocytosis appears immediately, no stage of leucopenia being obvious.

Septic infections, whether due to streptococci or staphylococci, are usually associated with a definite leucocytosis, the number of leucocytes being often increased as much as fourfold. Such diseases as erysipelas, cellulitis, and puerperal septicæmia come into this category, and also suppurative inflammations or abscesses in any part of the body.

That the presence of collections of pus may be diagnosed from the occurrence of leucocytosis is a fact of much clinical value. Thus Stengel has found, as the result of examination of the blood in a number of cases of appendicitis, that in those in which suppuration had supervened, in consequence of the presence of the staphylococcus albus, the number of leucocytes varied from 15,000 to 40,000; while in the non-suppurative cases leucocytosis was extremely moderate or absent.

Von Limbeck has indeed asserted that leucocytosis only appears when exudation into the tissues occurs, and that the extent of leucocytosis which accompanies it is directly dependent on the degree of cellular richness of the exudate. Thus leucocytosis is a usual concomitant of inflammations of serous membranes, whether of the peritoneum, pleura, or meninges. The extent of leucocytosis, however, often bears no relation to the amount of the exudation; and in tuberculous inflammations of serous membranes leucocytosis is invariably absent, unless in the case of secondary septic infection.

(iv.) *Leucocytosis of cachexia and malignant diseases.*—During the course of many cachectic diseases a leucocytosis of considerable extent is apt to arise. Stengel finds that this is particularly the case in congenital syphilis and rickets, the increase in the number of leucocytes being sometimes so great as to simulate leukæmia. In such cases the diagnosis is the more difficult as the increase in numbers mainly concerns the hyaline leucocytes.

In the moribund a *"terminal" leucocytosis* is frequently observed, especially in the subjects of protracted chronic disease. This is merely an exaggeration of the leucocytosis of cachexia. According to Cabot and others, this condition is specially apt to supervene in fatal cases of pernicious anæmia. The explanation of the condition is by no means simple; but it has been suggested that a terminal infection or a retardation of the circulation, with fall of blood-pressure, may bring about a discharge of leucocytes into the circulation.

The *leucocytosis of malignant disease* is believed by some authors to be due to the cachexia of the later stages. Others look upon the inflammation surrounding the focus of disease as the immediate cause. Leucocytosis is more pronounced in sarcoma than in carcinoma, and tends to be more marked the less circumscribed the growth.

(v.) *Post-hæmorrhagic leucocytosis.*—Loss of blood, especially if serious in amount, is rapidly followed by a leucocytosis, the extent of which is more or less dependent on that of the hæmorrhage on which it ensues. It may appear within an hour or so, and may persist for several days.

Observers differ somewhat as to the exact nature of the leucocytosis; some assert that the finely granular oxyphil (adult) cells are increased both absolutely and relatively, others that the most noticeable feature is an excess of the mononuclear hyaline corpuscles, and more particularly of the lymphocytes. It is not improbable, however, that these apparently divergent results may have relation to the cause of the hæmorrhage, whether traumatic or pathological, in each particular instance.

Concerning the actual causation of leucocytosis much difference of opinion exists. The current hypothesis is that the process mainly depends on what is named "chemotaxis." Thanks in large measure to the researches of Schulz, Von Limbeck, and of Goldscheider and Jacob abroad, together with those of Sherrington, and Hardy and Kanthack in this country, much light has recently been shed on the processes concerned in leucocytosis.

The mass of experimental evidence that has accumulated as the result of the labours of various workers in this field all tends to support the chemotactic hypothesis of leucocytosis, according to which leucocytosis must be regarded as in the main a phenomenon dependent on a chemical stimulus of a more or less intensive character, which is enabled to act on the hæmic leucocytes, and also on the blood-forming organs, through the medium of the circulating blood.

As previously stated, the injection of bacteria, or their metabolic products, whether toxins or proteins, or even simple traumatism, brings about, in the first instance, a rapid disappearance of leucocytes from the peripheral circulation.

This phase was thought by Löwit to be due to an actual destruction of corpuscles, to which process, therefore, he applied the name *leucolysis.*

This view is now no longer tenable since, as Schulz first indicated, the more or less complete disappearance of leucocytes from the peripheral circulation is due to an altered distribution; the leucopenia or hypoleucocytosis being coincident with a storage of leucocytes in the internal organs. That such is the case has been definitely proved by Goldscheider and Jacob, who found that the leucocytes accumulate chiefly in the capillaries of the lungs and liver.

This stage of hypoleucocytosis is due, as it appears, to a repellent action on the circulating leucocytes, and is in turn followed by a hyperleucocytosis or increase in the white corpuscles, provided that the injury inflicted on the organism be not of so severe a character as to render recovery improbable. This hyperleucocytosis may be due, as Schulz has suggested, to a secondary change in the distribution of the leucocytes, which, having been just previously packed away in the capillaries of certain of the viscera, now once again find their way back into the peripheral circulation, together with others which for the time are carried along with them, the increase in numbers above the normal being thus accounted for. The researches of Goldscheider and Jacob, however, afford reason to believe that this explanation is insufficient to account for the facts, and that at this stage the total number of leucocytes in the blood may be positively increased, although the place of origin of such additional supply may be difficult to determine.

Goldscheider and Jacob were able, indeed, to demonstrate that during this stage of hyperleucocytosis not only is the number of leucocytes in the capillary area of the pulmonary circulation equal to that of the leucopenic phase, but may be actually greater than before. It would seem fairly certain, therefore, that under the influence of chemotactic attraction the blood-forming organs are excited to greater activity, the result of which is seen in an abnormal output of cells which may happen to be stored at the time in these areas, together with simultaneous multiplication of leucocytes.

From experimental evidence we learn that the extent of the repellent action exerted on the hæmic leucocytes, as well as that of the subsequent leucocytosis, are determined, in large measure at any rate, by the virulence and the amount of the particular material injected. Thus the more potent the influence on the organism generally, the more pronounced will be not only the preliminary leucopenia, but also the secondary leucocytosis. It must be understood, however, that this statement only holds good up to a certain point; for when the dose and virulence of the noxious agent are sufficiently intense, and the consequent depression is so profound that the system is unable to rally, leucocytosis does not occur. It is possible, therefore, in most cases, to judge from the presence or absence of leucocytosis whether in any particular instance recovery will or will not take place—a sign which, as I have stated, has already been found of great assistance in the prognosis of specific infections in man.

In certain instances leucocytosis arises without any preliminary leucopenic phase. This occurrence has been described by Goldscheider and Jacob as a result of the experimental injection of a glycerine extract of spleen; and the same has been noted in animals which are either naturally or artificially immune.

II. ESTIMATION OF THE SPECIFIC GRAVITY OF THE BLOOD.—Until recently records of the specific gravity of the blood in disease have been very scanty, as it was necessary to remove a considerable quantity of blood for the purpose; moreover, the operation, involving as it usually did the use of the specific gravity bottle, was by no means an easy one. Moreover, by this method it was practically impossible, except with very elaborate precautions, to take the specific gravity of uncoagulated blood; hence defibrinated blood was used on the assumption that its specific gravity does not appreciably differ from that of the fluid circulating in the living vessels.

The ingenious method devised by the late Professor Roy, however, affords a means of making rapid and accurate observations at the expense of a single drop or even a fraction of a drop of blood.

Roy's method consists in observing whether a drop of the blood, rapidly withdrawn from the circulation and placed in a solution of known specific gravity, rises, sinks, or remains stationary in this solution.

Certain modifications of detail have been suggested by Dr. Lloyd Jones, and by myself, which have rendered it more easily applicable to clinical requirements.

The requisite apparatus consists of—

1. A series of solutions of various specific gravity, ranging from 1025 to 1070, one member of the series corresponding to each unit of the third place of decimals. For ordinary use, however, a much less number will suffice, as the numbers at the ends of the scale are seldom if ever required. In any case, however, for an extended series of observations, a considerable quantity of fluid corresponding to each degree employed should be provided. Roy originally used water to which glycerine was added in each case until the mixture was of the necessary specific gravity; but such solutions are apt to be untrustworthy, as the specific gravity is not constant, particularly if, as is often the case, a mould develop on the surface of the fluid. Fluids more suitable for the purpose may be made up from a stock solution of equal parts of glycerine and distilled water saturated with Barff's boro-glyceride and magnesium sulphate, with the addition of a small quantity of corrosive sublimate. If the specific gravity is to be lowered, this stock solution is diluted with water, and its density can be increased to any needful extent by the addition of more glycerine and boro-glyceride. Solutions thus made have a constant specific gravity. Moreover, blood does not clot very rapidly in them. The accuracy of the graduation should be ensured in the first instance by testing the specific gravity of the fluids with an accurate hydrometer, and by controlling these results with the balance. Small quantities of these fluids should be kept in a series of two-ounce bottles, the stoppers of which have been care-

fully greased so as to prevent any change of density by evaporation. The small bottles are best fitted into wooden stands for convenience of transit.

2. A number of glass pots about 1½ inch deep and half an inch wide, of which probably at least half a dozen will be required for one observation.

3. Fine capillary pipettes, formed by drawing out a piece of small glass tubing; the last quarter of an inch should be bent at right angles to the stem. To the opposite and wider extremity a piece of india-rubber tubing should be fixed, to which the mouth may be applied when the contents of the pipette are to be expelled.

It is usually possible, with practice, to make a fairly accurate guess at the specific gravity of the blood in each case, so that six or more of the small pots may be filled from the small bottles (by means of a fairly large pipette) with fluids of the densities likely to be required. Otherwise in a first observation every alternate number may be omitted so as to have a longer range at hand. A finger is then cleaned, and a fairly large drop of blood obtained by puncture with the precautions already laid down (p. 409). The drop is drawn by suction into one of the capillary pipettes, and, the pipette being lowered at once beneath the surface of the fluid in one of the small pots, some of the blood is gently blown into it. If the pipette be held so that the end is horizontal, the drop of blood expelled, if of the same density as the fluid contained in the pot, will have no tendency either to rise or to fall; if its specific gravity be higher than that of the fluid it will tend to fall, if lower to rise. With a little care it is comparatively easy to find the fluid in which it remains stationary, or at any rate to hit upon two adjoining numbers, in one of which it may slowly rise, and in the other slowly fall. To obtain a reading correct to a decimal part of a degree we shall carefully mix measured quantities of the two numbers between which the specific gravity has previously been found to lie, and in this manner readings to one-half or one-third of a degree, or even to one-tenth of a degree, may be obtained. It is worthy of note that the portion of the blood last expelled from the pipette is not infrequently some 0·0005 above that of the portion first expelled. This difference is due partly to capillary action in filling, and partly to friction of the blood against the wall of the pipette. If extreme accuracy be desired, the difficulty can be overcome by using the corresponding portion of each drop withdrawn.

In order to avoid any trace of admixture of two successive drops of blood, and to avoid the rapid clotting which goes on in the drop received into a pipette in which blood has previously been received, a fresh pipette, recently drawn in the blow-pipe flame, should be used for each observation. It is also necessary to see that the pipettes are carefully dried, as during their cooling moisture tends to condense in them.

Method of Hammerschlag.—This, which is essentially a modification of that originally devised by Roy, differs from the latter in that mixtures of chloroform and benzol are employed instead of more or less dilute glycerine solutions.

The supposed advantage of Hammerschlag's method is that a drop of blood when introduced into such a mixture as that devised by him does not tend to mix with it, but retains the appearance of a red bead.

Estimations are made in one of two ways: (α) A number of small pots are prepared, containing a series of mixtures of chloroform and benzol previously made up, and ranging in specific gravity from about 1035 to 1060. Into several of them in turn a drop of blood is introduced, by means of a bent capillary tube, until that mixture is found in which the drop of blood neither rises nor falls. (β) Chloroform and benzol are mixed in an ordinary urinometer glass in such proportions that, when tested by means of a urinometer possessing a somewhat extended scale of graduations, the specific gravity of the resulting mixture is found to be about that of normal blood (1055-1069). A drop of blood is then blown out into the mixture at a point beneath the surface by means of a bent capillary tube. If the bead tend to sink, chloroform is added drop by drop; if, on the contrary, the bead tend to rise, benzol is added in like manner. After every such addition the whole contents of the urinometer glass should be thoroughly stirred by means of a glass rod in order to ensure the uniformity of the specific gravity of the whole mixture. As soon as the drop of blood no longer shows any tendency either to rise or fall, the specific gravity of the surrounding liquid is obviously equal to that of the blood itself. All that now remains to be done is to take the specific gravity of the chloroform and benzol mixture by means of the urinometer, and the result thus obtained furnishes the required specific gravity of the specimen of blood.

Haycraft's method.—Two mixtures of benzyl chloride (sp. gr. 1100) and toluol (sp. gr. 0870·6) are made, the one (A) having a specific gravity of 1070, and the other (B) having a specific gravity of 1020. With a cubic centimetre pipette, graduated to $\frac{1}{100}$th c.c., one c.c. of A is measured off into a glass tube, and the drop of blood to be tested is then allowed also to flow into the tube. The drop of blood, having a different surface tension, does not mix with the solution, but floats on its surface as a tiny red globule. The graduated pipette is now filled with solution B, which is allowed to run slowly into the mixing tube, the tube being shaken after each addition. As B flows in, the specific gravity of the mixture falls, and after each addition and shaking the red globule returns more and more slowly to the surface. At last it tends neither to rise nor sink, and, the specific gravity of the mixture being now that of the blood itself, this can readily be calculated or read off from the table attached to the apparatus[1] sent out by the maker. Suppose 0·5 c.c. of B to have been added, the total weight of the fluid divided by its volume will give the specific gravity of the mixture:—

1 c.c. at sp. gr. 1070	1070
·5 ,, ,, 1020	510
	1·5)1580
	1053

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[1] Made by Mr. Fraser, Lothian Street, Edinburgh.

As the mixtures of benzyl chloride and toluol expand with heat they will vary in their specific gravity, so that, if exactitude be required, a correction for temperature must be made. The solutions A and B are prepared at the temperature of 15·6° centigrade, or 60° F., and if the temperature of the room be also 60° F. no correction will be needed. If, however, the surrounding temperature be higher than 60° F. the specific gravity of the fluids will be lowered, the fall of specific gravity being, roughly, at the rate of one degree for every 2° F.

In his original paper Haycraft warns those who may employ his method that it is well not to allow the fumes of benzyl chloride to get into the eyes, as, the vapour being very irritating, somewhat painful smarting may result.

Method of Schmaltz.—Mention must be made of this method because, although somewhat tedious, it gives very accurate results, and a large amount of work has been carried out by its means. As, however, it involves the use of delicate chemical balances it is hardly likely to come into general use in clinical work. The blood, of which the specific gravity is to be determined, is carefully weighed in a small capillary tube (pycnometer) of known weight. By subtraction the weight of the blood is obtained, and if this be divided by the weight of an equal amount of water the specific gravity of the blood is obtained.

Whatever the method, it is, of course, only the specific gravity of the blood, as a whole, which is thus determined. To what particular factor or factors, in each instance, alterations in the specific gravity of the blood are to be attributed remains to some extent a matter of conjecture. It is obvious that the alteration may be due to one or more of the following causes :—

1. An increase or diminution in the number of corpuscles in a given volume; the specific gravity of individual corpuscles, and of the plasma remaining unchanged.
2. An increase or diminution in the density of the plasma; the specific gravity and the number of corpuscles remaining unaltered.
3. A simultaneous increase or diminution in density both of corpuscles and plasma, with or without alteration in the number of corpuscles in a given volume of blood.

Schmaltz, from observations with his capillary pycnometer, concludes that, broadly speaking, the specific gravity of the blood varies directly as the percentage of hæmoglobin, but is largely independent of the number of red corpuscles. Hayem states that the specific gravity depends on the corpuscular richness of the blood—the difference possibly being caused by the passage of a certain amount of plasma into the lymph spaces. Dr. Lloyd Jones expresses somewhat the same opinion. Certain experiments of my own appear to show that, in the healthy animal at any rate, a rise of density of the blood, produced artificially, is accompanied by a somewhat closely corresponding rise in the number of red corpuscles.

These experiments further show that so long as the density remains unaltered, even under abnormal circumstances, the number of the corpuscles may also remain practically unaffected.

Again, in cases of paroxysmal hæmoglobinuria, when, during the paroxysm, the red corpuscles are broken up and the dissolved hæmoglobin has escaped from the blood, the diminution in number of the corpuscles is accompanied by a concurrent fall of the specific gravity of the blood; as the following observations show:—

	Specific Gravity of the Blood.	No. of Red Corpuscles.
Before paroxysm	1·0523	3,910,000
After ,,	1·0515	3,680,000
Before ,,	1·0575	3,710,000
After ,,	1·0505	3,440,000
Before ,,	1·0516	3,270,000
After ,,	1·0506	2,970,000
After ,,	1·0470	2,760,000

I have also shown that the specific gravity of the plasma itself usually falls concurrently with that of the total blood; this being especially the case after experimental injections into the blood-vessels, and in those cases in which the specific gravity of the blood as a whole has been lowered by hæmorrhage. It is also very noticeable in severe cases of pernicious anæmia. At the same time there can be no doubt that in the lowering of specific gravity which may occur under these various circumstances, the substance of the coloured corpuscles has its share of the additional amount of water, and that these corpuscles themselves also become of less specific gravity than previously.

If we desire to observe the specific gravity of the blood serum or plasma we may use Roy's method, the blood having previously been centrifuged in capillary tubes.

Professor Sherrington thus describes his method of obtaining the serum or plasma from small quantities of blood:—"A drop of blood, as it exudes from a prick in the skin, is taken by capillarity into a fine freshly-drawn glass tube, like a vaccine tube but longer, and bent into a U-shape. The capillary U-tube is then placed, with its bent end downwards, into a 'bucket' on the centrifuge, or on a radial slot on a vulcanite disc; the two open ends will then be toward the centre of rotation, and in a few minutes after the instrument is set in action a clear layer of serum or plasma is obtained in each limb of the tube."

Sherrington and myself have examined the specific gravity of the blood of a number of cases in well-marked anæmia; more than a hundred cases have been observed, with the results given in the appended table. Observations on other diseases have not been by any means so many, but the results are given for purposes of comparison. The observations were taken for the most part at the same time of day, about 11 A.M., a point which Lloyd Jones has shown to be of importance. For comparison certain results arrived at by Quincke and others, working with the older methods, are also brought together in the table, as well as some of those obtained by Lloyd Jones, who worked with Roy's method.

In the table the numbers on each side of a hyphen denote the maximum and minimum of the observations relating to the particular diseases; where one number only is given, this is the only one recorded.

Specific Gravity of the Blood in Various Diseases.

Disease.	Sherrington and Copeman.	Lloyd Jones.	Quincke.	Becquerel and Rodier.
Anæmia: Chlorosis[1]	1041–1043	1032–1045 (severe cases)	1035·2 – 1049·1 (probably included cases of pernicious anæmia)	1045·8 (mean of observations on six chlorotic girls)
Pernicious anæmia	1027–1034	1029–1040		
Leucocythæmia	1048·5–1051 (1 case in last stage) = 1032	...	1044·3	(1036 – 1049·5 Robertson) range of five cases
Gastric ulcer	1038 (very anæmic)–1050·5			
Lymphadenoma	1062			
Hæmoglobinuria	1047–1057			
Cardiac (none congenital)	1033–1052	Compensated 1054 Uncompensated 1051·6 Congenital 1061–1072	...	1052·5 (mean of series of 24 cases) 1050·2 (mean of series of 31 cases in third stage)
Diabetes	1058–1061	1054–1061	1054·9–1059·5	
Cirrhosis of liver with ascites	1046–1052	...	1049·6 (with hæmophilia)	
Acute nephritis	1041–1057	1038–1060		
Chronic nephritis	1054·5–1060	1034·5–1060	1047·3–1048·7	
Uræmia	1052	...	1050·5	
Tuberculosis (of kidney)	1048·5			
Tuberculous peritonitis	1057–1059			
Chorea	1050–1054			
Chronic hip disease	1042–1047			
Dysentery	1049–1052			
Chronic plumbism	1031			
Myxœdema	1058–1062			

It is worth noting that it is not possible in some cases to form a correct judgment of the probable specific gravity of the blood from the appearance of the patient, as, under certain circumstances at present but ill understood, the tint of the skin is not always indicative of the poorness or richness of the circulating blood. Oppenheimer, in the course of a series of observations on the enumeration of the blood corpuscles with the hæmocytometer, frequently came across such apparent discrepancies, which he attributes to irregular circulation.

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[1] Schmaltz, using his capillary pycnometer, found the blood in chlorosis possess a specific gravity of 1030–1049 (29 cases). But he apparently includes cases of pernicious anæmia.

Shock.—In the condition known as "*shock*," which is apt to supervene, to a greater or less degree, on serious injuries and on surgical operations—more particularly when the contents of the abdominal cavity are in any way interfered with—the specific gravity of the blood becomes raised, sometimes to a marked extent, as was first demonstrated experimentally by Sherrington and Copeman. This observation has since been abundantly corroborated by the result of the investigations of Grünbaum and others on the human subject. It is thus apparent that valuable information as to the condition of a patient subsequent to severe accidents or operations is obtainable by examination of the specific gravity of the blood. A well-marked rise of specific gravity, under the conditions indicated, is of distinctly unfavourable import. (See Art. "Shock and Collapse," vol. iii. p. 320.)

III. ESTIMATION OF THE COLOURING MATTER IN THE BLOOD.—Various methods have been elaborated from time to time for the estimation of hæmoglobin, but the colorimetric method is the only one which is sufficiently rapid for clinical purposes. Of the colorimetric instruments, devised for these estimations, those best known are Gowers' and v. Fleischl's. The latter apparatus is in general use on the Continent and in America, while, not unnaturally perhaps, the former is better known and more often employed in England. Although both these instruments require brief description, yet it is probable that they will be abandoned before long in favour of a more accurate hæmoglobinometer recently introduced by Dr. Oliver. In the colorimetric method a more or less diluted blood solution is compared with a colour standard which, in Oliver's and v. Fleischl's instruments, consists of tinted glass, and in that of Gowers of a glycerine-and-water solution of picric acid and picrocarmine solidified with gelatine. In the first two cases a definitely diluted blood is compared with standards of varying intensity, while in the third the blood solution is gradually diluted until its tint corresponds with that of the fixed standard. If any approach to accuracy in the results is to be looked for in the use of any of these instruments, we must provide—

(i.) A standard light.
(ii.) A reflecting surface of standard tint.
(iii.) A means of cutting off extraneous light.

(i.) The employment of a standard light is of special importance since the results obtained will vary with the nature, and, to some extent, with the intensity of the illumination. As it is practically impossible to ensure in every series of observations made by different persons that the source and intensity of the light employed shall be identical, it is desirable, in recording the results of hæmometric observations, that a note of the nature and position of the light employed should be appended. On this point Dr. Oliver strongly insisted in his Croonian lectures for 1896, in which he demonstrated, by means of curves, the extraordinary divergence in colour-value of progressive dilutions of blood when estimated, by means of Lovibond's graded glass colour standards, in daylight and by candle-light respectively.

The colour-curves drawn up by him are made by entering the colour units on the side of the diagram to form abscissæ with the standard gradations which appear at the foot. By daylight (Fig. 11) it will be observed that the colour-curves of the blood are made up of varying proportions of red, orange, and yellow. In the lower percentages (10, 20, 30, and 40) red does not exist as a separate colour; it is only present in combination with yellow, as an orange made up of equivalent proportions of each. In these lower gradations the place of red is taken by yellow.

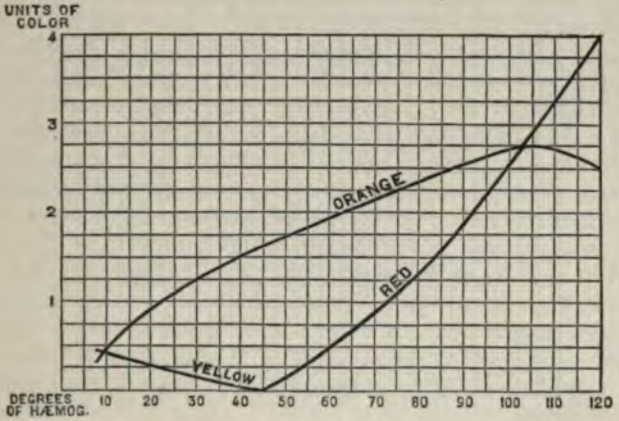

FIG. 11.—Specific colour-curve of blood, daylight (Oliver).

Between gradations 40 and 50 yellow dies out, and then red appears and increases progressively until at the highest grade it is the dominant colour. In these daylight gradations the curve of orange is the predominant one, though it begins to die out in the higher part of the scale. By candle-light (Fig. 12) the colour-curves are quite different and are less complicated.

Red is predominant throughout, except at the lowest grade, where it is subordinate to orange. From this point upwards orange gradually diminishes, and at 90 vanishes entirely; the highest grades are distinguished by pure red. The remarkable difference between the colour-curves furnished by the two kinds of light is doubtless due to the preponderance of yellow in the candle-flame.

Again, as Mr. Lovibond had previously observed in the matching of some of the aniline dyes, Dr. Oliver finds that the solution of blood possesses the quality of colour-purity, as distinct from colour-depth and colour-composition, or brilliancy in a remarkable degree; for it remains after these others have been duly matched. In the hæmoglobinometer devised by him this difficulty is met by using one of the lower grades of the blue glasses as a cover-glass to the blood-cell—an adjustment which does not disturb the correct reading of the hæmoglobin.

(ii.) Whatever source of light be employed the rays should be reflected from a "dead" surface of a pure white colour. In the instructions sent out with Gowers' hæmoglobinometer, it is suggested that the estimation should be made by holding the tubes between a white cloud and the eye of the observer; or that light should be reflected from a sheet of white paper held at an angle with the tubes. Sherrington, in measuring the amount of hæmoglobin in the blood by the Gowers' instrument, employs the light of a Welsbach lamp reflected from a vertical sheet of white paper not otherwise illuminated. To secure absolute uniformity of tint in the reflecting surface Lovibond uses pure, freshly precipitated calcium sulphate compressed into a slab. This material, which answers admirably, has been adopted in the construction of Oliver's and v. Fleischl's hæmoglobinometers.

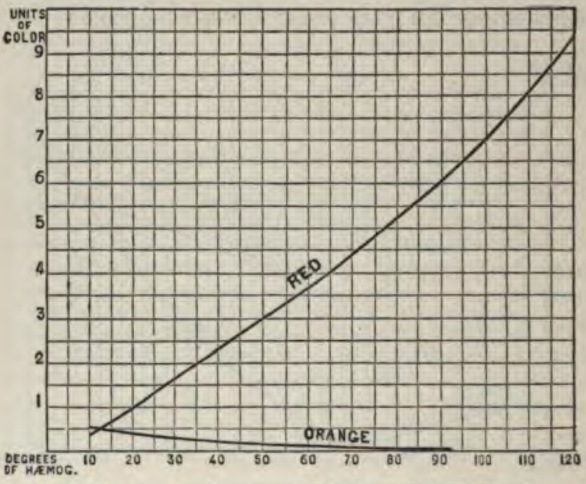

FIG. 12.—Specific colour-curve of blood, candle-light (Oliver).

(iii.) Increased accuracy in hæmometric observations may also be obtained by examining the blood solution and the colour standard through a tube of metal, or other material, of about ten inches in length and blackened within. The exclusion of outside light is thus ensured as well as the maintenance of a definite distance between the observing eye and the objects to be compared.

Oliver's hæmoglobinometer.—This apparatus is an adaptation to hæmometric work of the tintometer, an instrument invented some years ago by Mr. Lovibond of Salisbury for the purpose of estimating with scientific accuracy the true colour intensity of different substances, whether solid or liquid, which are employed in various manufacturing processes. As in the original instrument, use is made of tinted glass standards, with which the solution of blood, diluted to a definite degree, is compared by light reflected from a surface of pure calcium sulphate—the examination being made through a camera tube to exclude outside light.

With the tintometer which, before the introduction of Oliver's instrument, had been employed by myself for some years past in the estimation of the colour intensity of the blood, three sets of definitely graded glasses are provided; one for each of the dominant colours, red, yellow, and blue. The graduations in each set are of equivalent value, the test of which is the production of a neutral tint when equivalent grades of the three colours are mixed. On the other hand, any shade or degree of colour can be matched by appropriate combinations of non-equivalent grades, and the measurement thus made can be recorded numerically and so reproduced at will. By means of Lovibond's standards Oliver has determined the colour value of progressive dilutions of normal blood by uniform candle-light, and also by daylight. Corresponding to these values he provides a series of glass standards by comparison with which the percentage colour-intensity of any sample of blood can be read off at once. Oliver has done good service in so simplifying the use of the tintometer that, while his modification is much more compact and so more readily applicable to clinical work, all the essential points of the original instrument are retained. In the hæmoglobinometer, however, provision is made for utilising a double instead of a single transmission of light through the blood solution and the standard glasses. The originator of this method asserts that greater accuracy of estimation is rendered possible when such double transmission of light is utilised, especially if working with specimens which, as in the case of blood obtained from cases of severe anæmia, present a low colour intensity. On the other hand, when the colour more nearly approaches normal it is possible to carry out an estimation with half the quantity of blood that would otherwise be requisite.

The apparatus as sent out for use consists of—(i.) an automatic blood-measure; (ii.) a mixing pipette; (iii.) the blood-cell and cover-glass; (iv.) sets of standard gradations; (v.) riders; (vi.) the camera tube; (vii.) standard candles; (viii.) a bottle of antiseptic fluid, a lancet, needles, and thread.

(i.) The automatic blood measure has a capacity of 5 c.mm., and fills readily by capillary attraction. It is made of stout glass, and the end presented to the blood is well polished, so that all traces of blood can be removed from it by the finger. The bore is dried out before an observation by passing a needle through it threaded with darning cotton. The handle is useful for stirring together the blood and water in the blood-cell.

(ii.) The mixing pipette is provided with a rubber nozzle which fits over the polished end of the blood-measurer, and ensures the complete rinsing out of the blood with the first few drops of water.

(iii.) The blood-cell is of more than sufficient capacity to ensure the complete liberation of the hæmoglobin. When filled level with the rim it yields a blood solution of rather less than 1 per cent. It is itself the measure of the amount of water to be added, and it is quite easy to fill it accurately.

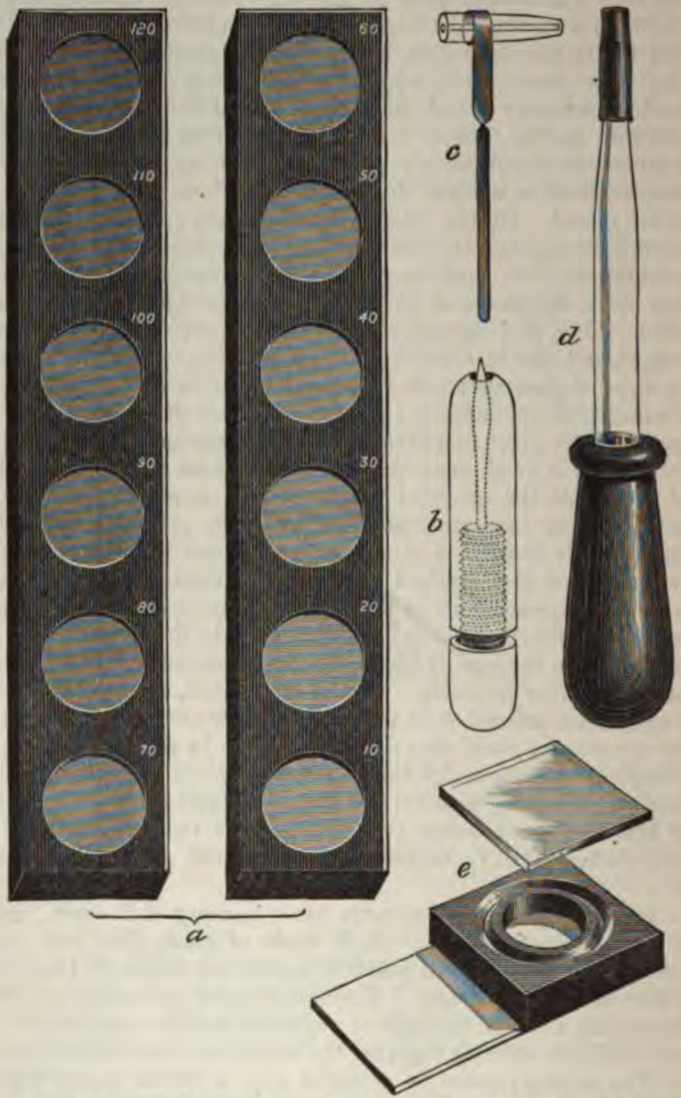

FIG. 13.—Oliver's hæmoglobinometer. *a*, Sets of standard colour grades; *b*, guarded lancet; *c*, automatic blood-measure; *d*, mixing pipette; *e*, blood-cell and cover-glass.

(iv.) The standard gradations are arranged as circular discs in two slabs, six in each; and they represent divisions of 10 degrees of the scale from 10 to 120 inclusive.

(v.) The riders are small squares of tinted glass provided for the reading of the degrees between each standard gradation. In use the slip of colourless glass is placed over the cover of the blood-cell, so as to balance the effect of the layer of glass of the rider which is laid over the standard. Two sets of riders have been arranged; one suitable to ensure the finer readings—for example, of 1 degree for physiological observation, and the other, sufficient for ordinary clinical observation, to enable the observer to determine differences of 2 degrees. For the reading of the units nine riders are required, which are grouped into three slides. It should be premised that the value of the riders is neither the same in the two standards required for candle-light and daylight, nor in the upper and lower halves of either standard. This want of uniformity arises, as previously stated, from the differences of the specific colour-curves in the two standards, and in the two portions of each. In the transition grades—the lowermost of the upper half and the uppermost of the lower half—this rule does not accurately apply; but inasmuch as the departure is constant in all observations, and is moreover slight, it may be disregarded for the sake of simplicity.

The daylight standard is less well adapted to the finer readings than the candle-light one, because the value of each rider becomes equal to 2 degrees in the upper half of the scale and 4 degrees in the lower half; and when candle-light is used each rider has an equivalent value in the six stronger grades of 1 degree and a double value in the six weaker grades of 2 degrees. For ordinary readings one rider only is used, namely, that which is equivalent to 5 degrees in each slab of the standards. Therefore each set of six standard gradations, whether for candle-light or daylight, has its own rider. When the blood solution is deeper in colour than any particular standard gradation, but is overstepped by the rider, the mean between the two may be taken as the reading; and the same rule will apply when the colour of the blood is higher than the rider, but is not so high as that of the next standard grade above. Hence this single rider may be made to provide readings of 0·25 and 0·75.

(vi.) The camera tube.—A tube of simple construction is provided which, being collapsible, will pack into a small compass with the other parts of the apparatus.

(vii.) The standard candles are of such a size as to afford a suitable and sufficiently uniform intensity of light. The position of the candle should be such as to furnish a high light, the flame being three or four inches above the cells. The observer will soon learn how to adjust the distance to the best advantage, so as to match the colours with the greatest certainty and accuracy. The actual distance does not affect the reading; but if the candle be placed too near, the glare becomes distractingly strong, especially when the lower grades, which require less light than the higher, are under observation.

The bore of the blood-measurer is first dried with the needle and cotton, and the polished point is presented to the drop of blood. The pipette must be quite filled, and if more than one application to the drop be needed, there must be no break in the column of blood. Any blood adhering to either end must be carefully wiped away with the finger. The rubber nozzle of the mixing pipette, charged with water, is now adjusted over the polished end of the pipette, and the blood washed into the blood-cell by pressing through the water drop by drop. The handle of the pipette is then used as a stirrer, and further additions of water, if required, are made so to impinge upon it as to graduate the size of the drops required to fill the cell accurately. It is easy to do this when the observer catches the reflection of a window on the surface of the fluid. A final thorough mixing with the handle will be required, and, to secure a level filling, another slight addition of water may be necessary. The cover-glass is then adjusted, when the presence of a small bubble signifies that the cell has not been overfilled. Finally, the blood-cell is placed by the side of the standard gradations, and the eye quickly recognises its approximate position on the scale. If the blood solution be matched in depth of colour by one of the standard grades the observation is at an end; but if it be higher

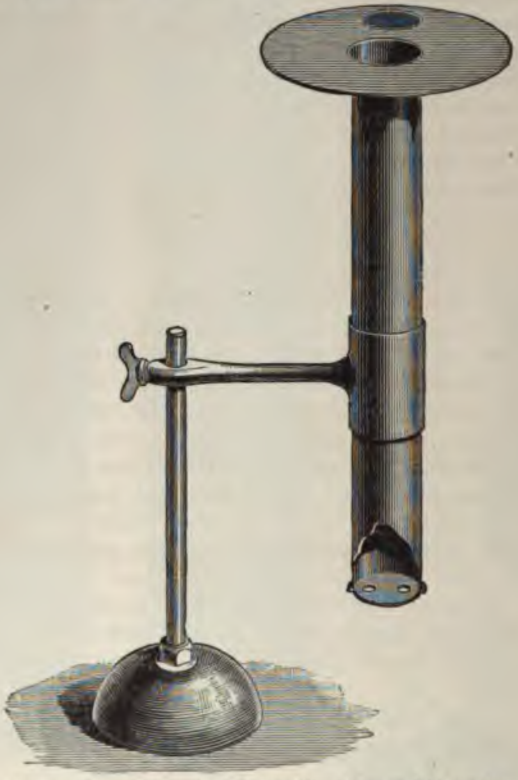

FIG. 14.—Camera tube for use with Oliver's hæmoglobinometer.

than one gradation, but lower than that above it, the blood-cell is placed opposite to the former and riders are added to complete the estimation. It is advisable to take a standard time, say ten seconds, for looking down the tube. If the eyes are strained with long working, it is well to look for a moment on the inside of the lid of the instrument case, which is lined with green morocco, complementary in colour to the blood and the colour-standards. This change rapidly restores the acuteness of observation.

In the more elaborate form of tube employed by Dr. Oliver the eyepiece is provided with a collar into which is let a piece of green glass. He finds that the most delicate appreciation of difference between the tints of the blood solution and of the standard is obtained by from time to time intercepting the impression of the discs by the finger, while the candle-flame is viewed for a few seconds through the green glass.

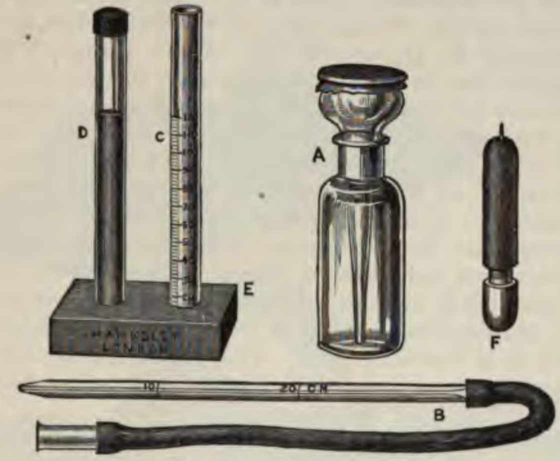

FIG. 15.—Gowers' hæmoglobinometer.

A, Bottle with pipette stopper for holding the diluting solution ; B, capillary pipette for measuring the blood ; C, graduated tube for measuring the amount of hæmoglobin ; D, standard tint of normal blood ; E, support for D and C ; F, puncturing needle.

Gowers' hæmoglobinometer.—This instrument consists of two small glass tubes of the same size. One contains a standard tint corresponding to a dilution of 20 cubic mm. of blood in 2 cubic centimetres of water (1 in 100); the standard is made of glycerine jelly tinted with carmine and picrocarmine. The second tube is graduated, so that 100 degrees are equal to 2 centimetres (100 times 20 cubic millimetres).

Twenty cubic millimetres of blood are measured by a capillary pipette (similar to, but larger than that used for his hæmocytometer), and after placing a few drops of distilled water in the second tube, this quantity of the blood is ejected into the bottom of it. The mixture is rapidly agitated by a rinsing action, and distilled water is then added, drop by drop, from the pipette stopper of a bottle supplied for that purpose, until the tint of the dilution matches that of the standard. The amount of added water indicates the amount of hæmoglobin present. As average normal blood yields the tint of the standard at 100 degrees of dilution, the number of degrees of dilution necessary to obtain the same tint with a given specimen of blood is the percentage proportion of the hæmoglobin contained in it. For instance, let 20 cubic millimetres of blood from an anæmic patient give the standard tint at 30 degrees of dilution, this specimen would contain only 30 per cent of the normal quantity of hæmoglobin. By ascertaining the corpuscular richness of the blood with the hæmocytometer we can compare the two. A fraction, of which the numerator is the percentage of corpuscles, gives at once the average value per corpuscle. Thus, if the blood containing 30 per cent of hæmoglobin contain 60 per cent of corpuscles, the average value of each corpuscle is $\frac{30}{60}$, or one-half of the normal. Sir William Gowers suggests that in using the instrument the tint be estimated by holding the tubes between the eye and a window, or by placing a piece of white paper behind the tubes. Care must be taken that the tubes are always held in the line of light, not below it, as in the latter case some light is reflected from suspended corpuscles from which the hæmoglobin has been dissolved. If all the light be transmitted directly through the tubes the corpuscles do not interfere with the tint. During 6 or 8 degrees of dilution it is difficult to distinguish a difference between the tint of the tubes; it is necessary, therefore, to note the degree at which the colour of the dilution ceases to be deeper than the standard, and also that at which it is distinctly paler. The degree midway between these two will represent the hæmoglobin percentage. The instrument is accurate within 2 or 3 per cent.

In order to obtain the greatest amount of accuracy in determinations of the hæmoglobin power of specimens of blood by means of Gowers' instrument, it is desirable not only to compare the tint of the contents of the two tubes by the aid of a standard artificial light reflected from a white surface, but also to cut off extraneous light as much as possible. With the old form of this instrument the writer finds that this may be effectually done by fixing an upright metal screen to the base in which the tubes are supported. This screen should have two narrow perpendicular slits corresponding to the central portion of the tubes; and it is well to have a movable slide of metal, working in the slits, which can be brought down level with the uppermost point at which the diluted blood stands in the graduated tube.

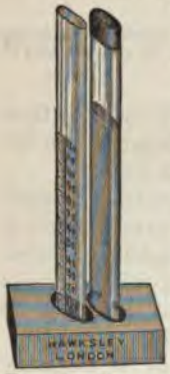

FIG. 16. — Gowers' hæmoglobinometer; improved form.

In the newer form recently brought out by Messrs. Hawksley an attempt is made to produce a similar effect by flattening the tubes, so that their contents present a more uniform tint from edge to edge, and by fixing the tubes in their support parallel to one another, but diagonally across the stand instead of side by side.

By looking at them in the proper position their adjoining edges appear to overlap, and as no white light is visible between the two tubes their respective tints can be more accurately compared.

v. Fleischl's hæmoglobinometer.—This instrument consists of a small metal stage, somewhat like that of a microscope, having on its upper surface a metal cylinder 1½ centimetres in length, which is open above and closed beneath by a glass plate, and is divided by a vertical metal partition into two equal parts. Beneath the stage a movable metal frame supports a long and narrow wedge-shaped slab of coloured glass, the colour ranging from deep red purple at the thickest end to clear glass at the opposite end.

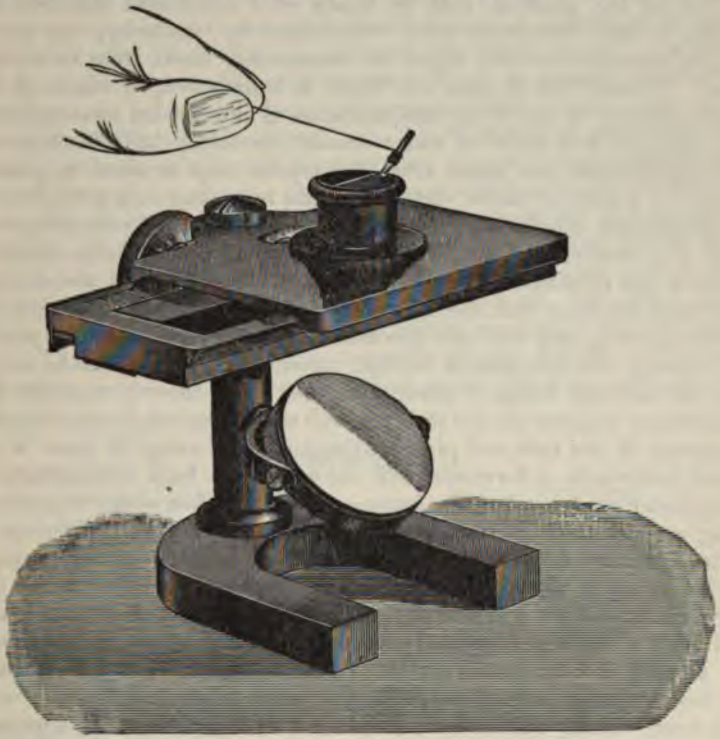

FIG. 17.—Von Fleischl's hæmometer.

The frame supporting the glass wedge moves on a rack and pinion attachment in a horizontal direction, so that every portion of the wedge can be brought in succession beneath the base of one of the cells formed by the divided metal tube. This portion of the cylinder is intended to be filled with water, the other half with diluted blood. By means of a reflector, the face of which is formed of a layer of pure calcium sulphate, light is directed upwards through the two cells. The source of light should always be a standard one, such as a Welsbach burner of known illuminating power—artificial light being, for several reasons, much better than daylight.

Capillary pipettes, for measuring the requisite amount of blood, are supplied with the instrument, their capacity being such that, when healthy blood is used, the colour of their contents on dilution to the requisite extent corresponds to that of the portion of the red glass wedge opposite the 100 graduation.

One of the pipettes, held by means of a short and flattened wire handle, is first completely filled with blood by capillarity, the pipette being afterwards carefully wiped to remove any blood from its sides or ends. Without loss of time the blood is transferred, by means of a to-and-fro motion, to one of the compartments of the divided metal cylinder, into which a few drops of water should previously have been poured. The expulsion of the blood from the pipette may be aided by pressing through it a drop or two of water by means of a glass tube provided with an indiarubber nozzle. Both compartments are now to be completely filled with water, that containing blood also being carefully stirred by means of the pipette handle, to ensure thorough mixture of the contents. Care must be taken that the fluid in one cell does not overflow into that in the adjoining cell, and that the metal cylinder is so placed in position on the stage that the base of the cell containing water is situated exactly over the coloured wedge of glass, while the light thrown upwards from the reflector reaches the eye through both compartments in equal amount. By means of the rack and pinion adjustment the wedge of glass is now moved backwards or forwards until the colours in both compartments of the cylinder correspond. The frame carrying the glass wedge is graduated along one side, and the number denoting the percentage of hæmoglobin in the specimen of blood under examination is read off directly through a small opening in the upper surface of the stage. It is by no means easy, however, to match the colour of the blood solution accurately, for reasons that have already been stated; and this is particularly the case when the amount of hæmoglobin in the blood is small. The graduation of the instrument is also somewhat inaccurate, percentages of 80 or 90 only being usually shown in examination of blood which is apparently normal. Cabot recommends the observer to look at the "divided cylinder" from one side, so that the image of the two cells shall fall on the lateral instead of on the upper and lower portions of the retina; under which circumstances, he says, a more correct judgment is possible. He further advises that the source of light should be placed at such a distance from the instrument as to reduce the intensity to a point barely sufficient for the estimation. Comparison of the colour value of the blood solution with that of the glass wedge will also be easier if both be observed through a tube or roll of paper blackened on the inside.

IV. Enumeration of the blood corpuscles.—This method consists in the dilution of the blood to a considerable but known extent, and the subsequent counting, under the microscope, of the number of the corpuscles in a small and measured amount of such diluted blood. A comparatively simple calculation will then enable us to estimate the number present in any given bulk of the blood originally taken for our purpose. In both of the modes, which will be described in detail, the corpuscles in the small sample of diluted blood are reckoned by means of a series of micrometer squares ruled over a certain area of the glass floor of the chamber or cell.

In Gowers' hæmocytometer, the instrument which up to the present has been in most general use in England, measured quantities of blood and of the appropriate diluting solution are drawn up in a couple of pipettes of known capacity, the mixture being afterwards effected by blowing out the contents of each of the pipettes into a small glass pot in which they are thoroughly stirred. In the Thoma-Zeiss instrument one pipette serves not only for the measurement both of blood and diluting solution, but also for ensuring the subsequent admixture of one with the other. The exact composition of the fluid employed for dilution of the blood is to some extent a matter of indifference, provided that it be of such a nature as not to act injuriously on the corpuscles, and of such specific gravity that the corpuscles readily sink in it. A normal saline solution (NaCl 0·7 per cent in distilled water) serves the purpose; or a solution of sulphate of soda having a specific gravity of 1025, as suggested by Gowers, may be employed. Other useful solutions are the following:—

Hayem's Fluid.

Mercuric bichloride . . .	0·25 gram.
Sodium chloride	0·5 „
Sodium sulphate . . .	2·5 grams.
Distilled water	100·0 c.c.

Thoma's Fluid.

Acetic acid	0·3-0·5 c.c.
Distilled water	100 c.c.

Recommended by Thoma as useful in the determination of the number of leucocytes, the red corpuscles being dissolved by the acetic acid.

Toisson's Fluid.

Methyl violet	0·025 gram.
Sodium chloride	1·0 „
Sodium sulphate . . .	8·0 grams.
Glycerine	30·0 c.c.
Distilled water	160·0 c.c.

The addition of the methyl violet or other aniline dye facilitates the counting of the white corpuscles by staining them, and so rendering them more conspicuous.

Sherrington's Fluid.

Ehrlich's purified methylene blue	0·1 grm.
Sodium chloride	1·2 ,,
Neutral potassium oxalate	1·2 ,,
Distilled water	300·0 c.c.

In this solution both chromocytes and leucocytes may be studied for an almost indefinite length of time without losing their characteristic appearances, especially if the examination be carried out on the warm stage.

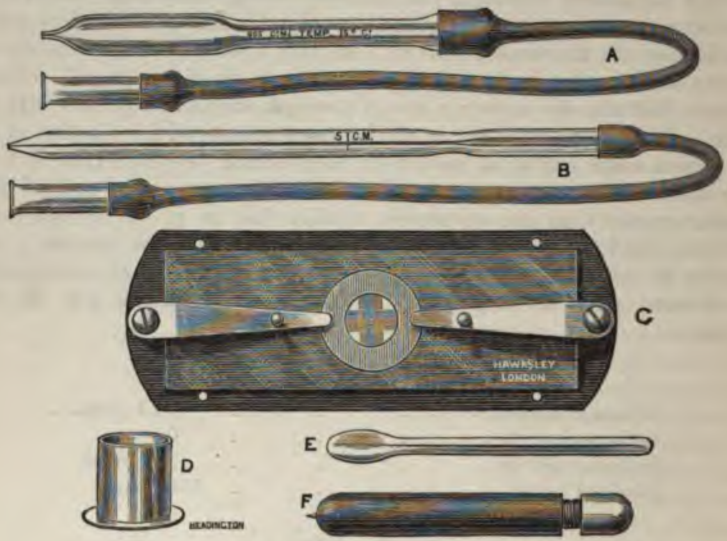

FIG. 18.—Gowers' hæmocytometer.

A, Pipette for measuring the diluting solution; B, capillary tube for measuring the blood; C, cell with divisions on the floor, mounted on a slide, to which springs are fixed to secure the cover-glass; D, vessel in which the solution is made; E, spud for mixing the blood and solution; F, guarded spear-pointed needle.

Gowers' hæmocytometer.—This instrument, a modification by Sir W. R. Gowers of that originally devised by MM. Hayem and Nachet, consists of (*a*) a small pipette, which, when filled to the mark on its stem, holds exactly 995 cubic millimetres—this pipette is furnished with an indiarubber tube and mouthpiece to facilitate filling and emptying; (*b*) a capillary tube marked to contain exactly 5 cubic millimetres; also filled with an indiarubber tube and mouthpiece; (*c*) a small glass jar in which the dilution is made; (*d*) a glass stirrer for mixing the blood and solution in the glass jar; (*e*) a brass stage plate, carrying a glass slip on which is a cell one-fifth of a millimetre deep. The floor of this cell is divided by ruled lines into one-tenth millimetre squares. Upon the top of the cell when in use rests a cover-glass, which is kept in its place by the pressure of two springs fixed to uprights at each end of the stage plate.

The instrument is employed as follows:—995 cubic millimetres of the solution are first placed in the mixing jar, after which 5 cubic millimetres of blood are drawn into the capillary tube from a puncture in the finger, and then blown into the solution. The two fluids are well mixed by rotating the stirrer between the thumb and the finger, and a small drop of this diluted blood is placed in the centre of the cell. The cover-glass is now gently lowered upon the cell and secured by the two springs, after which the plate is placed upon the stage of the microscope. The lens is then focussed for the squares. In a few minutes the corpuscles will have sunk to the bottom of the cell, and are seen at rest on the squares. The number is then counted, and the cubic area of diluted blood over each square is of such amount that this multiplied by 10,000 gives the number in a cubic millimetre of blood. Taking 5,000,000 corpuscles as the average per cubic millimetre for healthy blood, the average number in two squares of the cell is 100. Consequently the number of corpuscles present in two squares (ascertained by counting a larger number, such as 10 or 20, and taking the mean) expresses the percentage proportion of the corpuscles in the specimen of blood under observation to that of health; or, made into a two-place decimal, the proportion which the corpuscular richness of the specimen bears to that of normal blood taken as unity. In making such examination a sufficiently large drop of blood must be obtained by puncture with the point of the lancet without the employment of much pressure, for if the finger be much pressed or squeezed or ligatured, the relative amount of serum and corpuscles contained in the sample of blood abstracted is liable to be affected, and thus to afford inaccurate and consequently valueless results. It is somewhat difficult to draw the exact quantity of blood into the capillary tube, because in removing the blood from its point a little is easily drawn out of the tube; it is therefore better to draw rather more than the required quantity into the tube, then to remove the blood from the point with a soft cloth, and keep the cloth in contact with the point while the extra blood is blown out. A little of the diluting fluid should be drawn into the tube after the blood is ejected to ensure the removal of all the corpuscles. The smaller end of the stirrer may be used to remove the drop of diluted blood from the mixer to the cell. This drop must be deposited in the middle of the cell over the ruled squares, and care must be taken not to rub the stirrer on the engraved portion of the floor. The lines which form the divisions of the micrometer cell may be made more distinct in the following manner:—With a sharp penknife scrape a little of the lead of an ordinary soft lead pencil so that it falls into the middle of the micrometer cell, then, with a clean dry finger-tip, or a knot tied in a small piece of chamois leather, rub the powdered lead well over the cell; it may then be wiped in the ordinary way, and any of the lead which remains in the corners of the cell easily dusted away with a camel-hair brush. Powdered carmine may be employed in a similar fashion.

The cover-glass should be held in a horizontal position as it is being lowered on to the cell. When this has been done, the drop of solution should appear as a disk as nearly as possible in the middle of the cell, and care must be taken that the fluid does not run to the sides of the chamber. The two springs are to be carefully placed opposite each other by lifting them on the cover-glass, and not by "sliding" them.

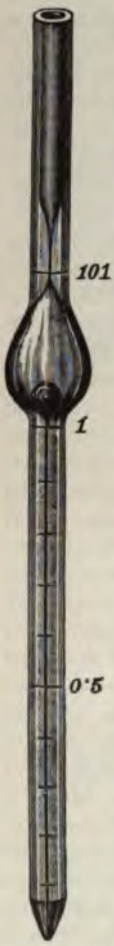

Fig. 19.—Capillary tube (Thoma-Zeiss apparatus for counting the blood corpuscles).

Gowers advises that the corpuscles should be counted in or near to the centre of the drop, and says that by raising the objective out of focus the white cells may readily be distinguished by their greater refractive power. A light which should not be too intense, and the position of which is so arranged that the rays when reflected upwards pass diagonally across the squares, will give the best illumination for both sets of lines. The whole process from beginning to end need not occupy more than a quarter of an hour.

The blood should not be drawn until the diluting solution has been placed in the mixing jar and the capillary tube is ready for use. The blood should be expelled immediately, as otherwise it may coagulate. Immediately after use, the pipette and capillary tube should have clean water drawn up into them; this may be followed by alcohol and ether if rapid drying be necessary. If either be obstructed, a horse hair or a piece of fine brass wire will probably clear it; or it may be cleared by placing it in nitric acid, all trace of which must afterwards be removed with water. In standardising the pipettes, the residual fluid, that which unavoidably adheres to the inner walls of the tubes, has been allowed for, so that the quantity ejected is exactly that indicated by the division upon each tube.

The cell must be cleansed after each observation by means of a small camel-hair brush and some clean water, after which it is dried carefully with a soft cloth. The cell must never be used a second time whilst damp, or the globule of fluid will at once disperse over its surface, and the corpuscles will not be deposited evenly over the floor.

The **Thoma-Zeiss hæmocytometer** is in most general use for the enumeration of the blood corpuscles on the Continent and in America. As in Gowers' hæmocytometer, the essential parts of the instrument consist of a counting-chamber and of a measuring pipette, which serves the double purpose of taking up the required amount of blood and of enabling it to be mixed with a definite quantity of the diluting fluid. The counting-chamber is formed of a glass slide supporting a square glass cell with a central circular aperture. Within this is fixed a smaller disc of glass ruled on its upper surface into a series of microscopic squares; the thickness of the disc being such that, when an accurately ground cover-glass is lowered over the aperture of the square cell, an interval of exactly 0·1 millimetre is left between the adjoining faces of cover-glass and disc.

The little moat which separates the internal edge of the cell wall and the periphery of the disc serves to catch any excess of fluid for which there may not be room in the space between the disc and the cover-glass. The size of each square is the $\frac{1}{400}$ of a square millimetre, the individual lines being exactly $\frac{1}{20}$ of a millimetre apart. The area over each ruled square has then the value of $\frac{1}{4000}$ of a cubic millimetre. The small squares are marked into groups of sixteen by means of more thickly ruled lines; these larger squares being very useful in reckoning the white corpuscles.

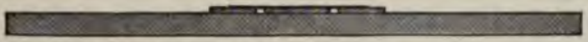

FIG. 20.

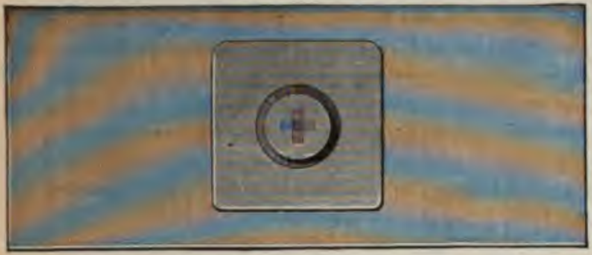

FIG. 21

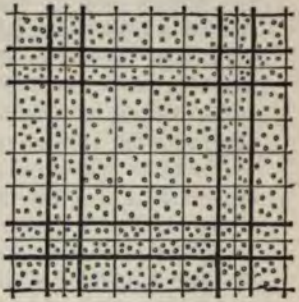

FIG. 22.

Thoma-Zeiss apparatus for counting blood corpuscles.

The diluting pipette consists of a thick-walled capillary tube which towards its upper end expands into a bulb, above which the pipette has again its capillary bore. Contained within the bulb is a small bead of glass for the purpose of facilitating the admixture of blood with the diluting solution. The lower extremity of the pipette is bevelled to a point and polished so that any excess of blood, or other fluid, clinging to it can easily be wiped off with a soft cloth. The portion of the pipette below the bulb is ruled off by cross lines marking tenths from 0·1 to 0·5 and 1; while above the bulb the mark 101 is found.

When using the apparatus the point of the pipette is applied to a drop of blood obtained in the manner previously described. By gentle suction, through an indiarubber tube attached to the upper portion of the pipette, blood is drawn up to the 0·5 or the 1 mark; after which the diluting solution is also drawn up until the mark 101 is reached, by which a dilution of the blood of 1 in 100 or 1 in 200 is obtained, according to whether the half or the whole of the capillary portion of the pipette has been filled with blood. The pipette is then gently rotated in the fingers for about a minute in order to ensure thorough admixture of the contents of the bulb, the process being aided by the presence of the little bead of glass. It is a matter of some little difficulty to draw up into the pipette exactly the amount of blood required. For this reason it is well to employ the dilution of 1 in 200, as in case the amount of blood obtained at the first attempt should reach slightly above the 0·5 graduation, the quantity in excess can be got rid of again by gently blowing through the tube; whereas if the higher mark should be exceeded and blood escape into the bulb it is useless to continue the operation until the whole pipette has been most carefully cleaned out and dried. Moreover, if the blood be of fairly normal quality, the number of red corpuscles in it will be so crowded over the surface of the ruled squares as seriously to interfere with the ease and accuracy of the counting.

Professor Sherrington finds that the greatest amount of accuracy in the enumeration of the blood corpuscles is to be obtained by the use of the Abbé-Zeiss counting-chamber in conjunction with the pipette made by Hawksley, instead of the one (devised by Thoma) which is usually sent out with the instrument.

According to Sherrington, the objections to the Thoma pipette are the large internal surface relatively to cubic content, the difficulty of drying the bead in the mixing-chamber quickly enough for use in successive observations, and the presumption that leucocytes will adhere to the bead. These objections are obviated in the new form of pipette containing no bead, and possessing a wider bore than the Thoma instrument. Sherrington also lays much stress on the importance of counting both chromocytes and leucocytes in the same film of the diluted blood; and of enumerating in the same film the representatives of the various leucocytes distinguishable when this step is considered necessary. And there can be no doubt that this plan is much preferable to that of counting the chromocytes in one film, the leucocytes generally in a second, and of determining the numerical proportion of their different varieties in yet another. It must, however, be borne in mind that it may not be possible to obtain Ehrlich's colour reactions with a living film.

When counting the leucocytes with the Thoma-Zeiss apparatus it is usual to dilute the blood ten times only; under which conditions the chromocytes must be rendered invisible in some manner, as otherwise their number tends to obscure the leucocytes. This difficulty may be overcome by employing Thoma's 0·3 acetic acid solution for diluting the blood; this having the effect of "laking" the chromocytes.

After a time, however, the leucocytes are also affected by the acid, so that it is better to dilute the blood to a larger extent and to enumerate both chromocytes and leucocytes at the same time. With care and practice this can be done if the dilution is carried out in the proportion of forty-nine parts of solution to one part of blood. In order to render the leucocytes more obvious I have been accustomed for many years past to dilute the blood with normal saline solution just tinged with a few drops of a watery solution of some aniline dye; but for this purpose the mixture devised by Sherrington (p. 442) is preferable, as the blood corpuscles remain entirely unaltered in it for considerable periods.

When the leucocytes are enumerated in a fifty-times diluted blood, as suggested, it is well to obtain an additional basis for calculation, not only by counting those lying on the squares, but also those over the whole of the floor space as far as the ruled lines extend. The area of this enlarged space must previously have been calculated, as it varies somewhat with each instrument.

V. ESTIMATION OF THE REACTION OF THE BLOOD.—Under normal conditions the reaction of the blood, as tested by litmus, is invariably alkaline. Slight variations in the degree of alkalinity can indeed be demonstrated in connection with such physiological processes as the digestion of food, in which the alkalinity of the blood tends to become increased; or as the after-effects of severe muscular exertion, by which, on the other hand, it is for a time somewhat diminished. Even under pathological conditions, however, it is unusual to find any extreme departure from the normal range; and although such variations as have been observed in the course of those diseases in which systematic investigation of the blood reactions have been carried out, show that change is usually in the direction of a fall of alkalinity, it is doubtful whether an acid reaction has ever been demonstrated during life. It is a somewhat curious fact that the reaction of the blood is, for the most part at any rate, dependent on the presence in it of sodium hydrogen carbonate ($NaHCO_3$) and sodium hydrogen phosphate (NO_2HPO_4), both of which, chemically speaking, are acid salts. As, however, these salts are formed of bases very loosely combined with acids, they are readily dissociated when brought into connection with a substance, such as litmus, which is capable of withdrawing the bases and uniting with them to form a distinctively coloured salt; this colour production serves, therefore, as a test not only for free bases, but also for bases which are combined with weak acids. This latter power is not possessed by certain other substances which are sometimes employed as indicators in alkalimetric investigations, such for instance as phenolphthalein; for this reason they cannot be employed in observations on the reaction of the blood. Fortunately, however, we have in carefully prepared red or neutral litmus an indicator sufficiently delicate (according to the observations of Wright and others) to show an immediate and distinct colour change with normal human serum which has been diluted as much as forty-fold. In all the various methods that have been proposed for the estimation of the reaction of the blood litmus has been employed as an indicator. In the earlier qualitative methods of Leibreich and Schäfer, slabs of plaster of Paris in the one case, and sheets of glazed paper in the other, impregnated with litmus were used, the intention being to retain the blood corpuscles on the surface, from which they could afterwards be washed in order to bring any colour-change into view; meanwhile the fluid portion of the blood, soaking into the substance of the slab or paper, was thus enabled to react with the previously absorbed litmus.

If it be desired to estimate *quantitatively* the degree of alkalinity of the blood, it becomes necessary to employ not only an indicator, but also a standardised solution of some acid. In the titration methods first introduced phosphoric acid was employed for this purpose by Zuntz, tartaric acid by Lasser and Landois, oxalic acid by Drouin. Although convenient to prepare, solutions of these organic acids tend to weaken after a time as the result of exposure to light and air; for this reason they have been discarded in favour of suitably dilute solutions of sulphuric acid. Landois' method, as improved by Drouin, is probably that which has been most extensively employed; but it possesses the serious disadvantage that, in order to obviate a difficulty introduced by the presence of the red corpuscles, the blood has to be considerably diluted with solutions of sulphate of soda, by which the estimation of the exact neutral point is rendered somewhat difficult. This objection cannot be advanced against the methods now to be described.

Haycraft and Williamson's method.[1]—This method is a development of the qualitative test originally introduced by Schäfer. The alkalinity is estimated by a graduated series of red litmus-papers.

To *prepare the papers*, place over-night a dozen half-sheets of cream-laid notepaper under a tap of running water in order to remove the acid generally present. Soak the papers in strong neutral litmus and dry them. Neutral litmus-papers may be purchased if desired. Pour a pint of normal (7 c.c. per 1000 c.c. of water) sulphuric acid into a shallow basin, plunge into it for half a minute a sheet of litmus-paper, withdraw it, blot it, and dry it; this is the strongest acid paper.

Now dilute the normal sulphuric acid with an equal volume of water, soak another sheet of litmus-paper, blot, dry, and mark it. Dilute the acid again and again until eight or ten papers are prepared. These should dry in the horizontal plane so that the acid does not gravitate to one border of the paper.

When prepared, the papers must be glazed by passing them between steel rollers. Any large stationer will do this, and the papers are then ready for use. Each sheet may be cut up into strips, or these may be cut off as required.

The *method* is to cleanse the finger of the patient and to puncture it with a broad-tipped stilette; the blood must not be squeezed from the finger. A paper, say D, is brought in contact with the drop for ten seconds, and then dipped in water. If there is no blue stain try E, and if there is a blue stain try C. The operator will soon find out the paper which just gives a reaction with normal blood, and he will be able in other cases to judge roughly if deviations from the normal are present. This method does not pretend to great exactitude, and may be classed in this respect with some other clinical methods, such as the estimation of chlorides in urine by the subsidence of the precipitate.

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[1] Communicated by Professor Haycraft.

Undoubtedly Haycraft's method possesses the merit of simplicity, but it has undergone severe criticism at the hands of Hutchison, who has stated that the results afforded by it are apt to show an extraordinary divergence from those obtained by means of the titration method. Thus he finds that the alkalinity of the blood in anæmia, as tested by the method of Haycraft and Williamson, is invariably above normal; and often, apparently, to an excessive degree. On the other hand, when the titration method is employed it is, according to this observer, as invariably found that the alkalinity of the blood is reduced. Hutchison explains this apparent contradiction between the results afforded by the two methods on the ground that the alkalinity of the plasma alone is ascertained by the glazed litmus-paper method, the alkali contained in the corpuscles which are left on the surface of the paper not being estimated, as in the titration method, during the performance of which the breaking down of the corpuscles liberates their contained alkali. Moreover, Hutchison concludes, as the results of experiments, that the more watery the blood the more readily the plasma percolates into the glazed litmus-paper. Under these circumstances the contained acid is more readily neutralised, and a fictitious value is given to the amount of alkali present in the blood. This difficulty cannot be surmounted by allowing the drop of blood to remain for a longer time in contact with the paper, for, as all observers are agreed, the alkalinity of the blood diminishes rapidly after its removal from the body. But the titration method of Landois, which, according to Hutchison, affords an estimate of the total amount of alkali in the blood, requires considerable care in its performance, if this result is to be obtained; since, as Loewy has shown, it is only when the blood is titrated very slowly at body temperature that all the corpuscles are broken down.

With reference to this point, however, it has always appeared to me that since it is the plasma of the blood and not the corpuscles that come into direct relation with the tissues, it is the estimation of the alkalinity of the plasma rather than that of the total blood which is of more immediate interest from the clinical point of view. I have been accustomed, therefore, for the purpose of observations on the reaction of the blood, to employ specimens of plasma (or serum) obtained by centrifuging a few drops of blood in capillary U-shaped tubes, after the manner originally employed by Sherrington and myself when working at the subject of specific gravity. Treated in this manner, a few minutes suffice to separate entirely the corpuscles from the plasma (or serum), portions of which taken up and measured in capillary tubes should then be mixed with exactly similar quantities of varying dilutions of normal sulphuric acid; the reaction in each case being tested by means of sensitive litmus-paper.

Wright's method.—This is a titration method also in which, as is now almost invariably the case, litmus is used as the indicator, and normal sulphuric acid, in appropriate dilutions, to neutralise the alkali of the blood. Unlike Hutchison, however, Prof. Wright maintains that, as the result of his own observations, as also of those of Drouin, "changes in the alkalinity of the circulating blood invariably manifest themselves in changes of the alkalinity of the serum." Acting on this assumption, therefore, he prefers for hæmalkalimetric observations to employ serum, and preferably that which has exuded from a blood-clot, rather than fresh plasma "contaminated" with red blood corpuscles.

As is, however, agreed on all hands, the alkalinity of blood undergoes a gradual diminution after removal from the living vessels; and consequently Wright thinks it well to postpone the estimation for some hours until, as he believes, a condition of stable equilibrium is reached.

It will be obvious, therefore, that the results obtained by him, although they may be comparable among themselves, do not afford an accurate estimate of the alkalinity of the freshly-drawn blood. On the other hand, if, as I have suggested, the blood be centrifuged, an estimation can be made within a few minutes of its withdrawal; and, in the absence of red corpuscles, the fall of alkalinity, if indeed it occur, is at anyrate much less rapid than is otherwise the case, and so may be neglected.

In Wright's method five progressive dilutions—twenty, thirty, forty, fifty, and sixty-fold—of normal sulphuric acid are employed in the titration.

This is performed by first drawing up into a fine capillary tube about one-sixth of the amount of serum available, followed by an equal amount of dilute acid. Accurate measurement is ensured by marking the pipette at the point reached by the serum, tilting it so as to include a bubble of air in the bore, and finally filling it up to the original mark with the acid solution. The exactly equal amounts of serum and acid thus obtained are next blown out into a watch-glass, thoroughly mixed and tested by transferring a series of separate drops to the surface of a strip of red litmus-paper.

If the twenty-fold diluted acid solution has been employed, it will probably be found that in working with normal blood the mixture will show an excess of acid. In this case it will be necessary to proceed, in precisely similar manner, to titrate with each other equal volumes of serum and the thirty-fold diluted solution. Intermediate degrees of alkalinity can be estimated by mixing, in a clean watch-glass, equal volumes of, say, thirty and forty-fold diluted normal acid, and titrating with the resulting thirty-five-fold acid solution. If this dilution should suffice to neutralise the acidity of the given sample of serum exactly, the result is expressed by the fraction $\frac{N}{35}$. Prof. Wright found, as the result of a number of estimations of apparently normal blood, that the serum has an alkalinity which varies between the values $\frac{N}{30}$ and $\frac{N}{45}$, the average being about $\frac{N}{35}$.

It has been shown that during health the constancy of the level at which the alkalinity of the blood is maintained is so great as to suggest that some regulating mechanism must be continuously at work to secure it. And in many diseased conditions the action of this regulating mechanism is disturbed in so slight a degree that no appreciable departure from the normal slight phasic variations is demonstrable. Under certain circumstances, however, marked alterations may be found; this being specially so in the specific fevers, in the various forms of anæmia, including leukæmia, in diabetes, particularly if coma be about to supervene, in uræmia, gout, and jaundice, and in certain cases of poisoning, as for instance by carbonic oxide or potassium chlorate. The alteration that occurs under these conditions is almost invariably in the direction of a lowering of alkalinity, due, as it would appear, to the presence in the blood of various acids, such as lactic, uric, oxybutyric or bile acids respectively, according to the particular disease under consideration. In anæmia, however, and markedly in chlorosis, an increase of alkalinity is usually present, this being the more obvious if it be the reaction of the blood plasma which is estimated rather than that of the total blood.

VI. Determination of the coagulation time of the blood.—When first shed, blood appears to the naked eye as a homogeneous red fluid; but at a longer or shorter interval, after removal from the body, it gradually separates into two portions, consisting of a semi-solid coagulum or clot, which still remains red—though the colour gradually becomes somewhat darker—and a clear straw-coloured fluid, the serum, the amount of the latter gradually increasing as more of it is squeezed out of the interstices of the contracting clot.

The clot consists of a fine meshwork of interlacing filaments of fibrin; the red colour and the chief bulk of the clot being due to the entanglement of the red blood corpuscles amid the threads of fibrin. Fibrin may be obtained free from corpuscles by washing the clot thoroughly under a stream of water until the washings are no longer coloured: the grayish-white, stringy mass which remains is fairly pure fibrin. If coagulation take place more slowly than usual the corpuscles will have time, before the separation of the serum, to sink towards the lower portion of the vessel into which the blood is received; and in such case the upper layer of the clot which eventually appears will be more or less devoid of colour: this portion is what is known as the "buffy coat."

In certain pathological states, such as hæmophilia for instance, the rate of coagulation is important both in respect of the disease itself and of the results of treatment. For clinical purposes the "coagulation time" can be ascertained by allowing a few drops of blood from a finger-prick to fall on a glass slide, taking care that the individual drops remain isolated from one another, and are fairly equal in size. If now a fine and carefully cleansed needle be drawn through one drop after the other at short but regular intervals, the moment at which coagulation begins will be found by observing the lapse of time between the reception of the drops of blood upon the glass slide and the drawing out of a filament of fibrin from a drop by the needle.

More accurate information may be obtained by means of the instruments devised by Professor Wright and by Drs. Brodie and Russell. In Wright's method the blood coagulability is determined by aspirating blood into a series of tubes, and by then blowing down tube after tube in succession until coagulation takes place; when of course blood can no longer be blown out of a tube. The lapse of time since the blood was shed is known as the "coagulation time." The necessary apparatus, as seen by reference to the figure, consists of a water-tin surrounded by a leather jacket lined with flannel, and constructed with a series of pockets between the layers of flannel; each pocket being just sufficiently large to admit a coagulation tube. One of the pockets is reserved for a glass thermometer comparable in diameter with the coagulation tubes in the remaining pockets.

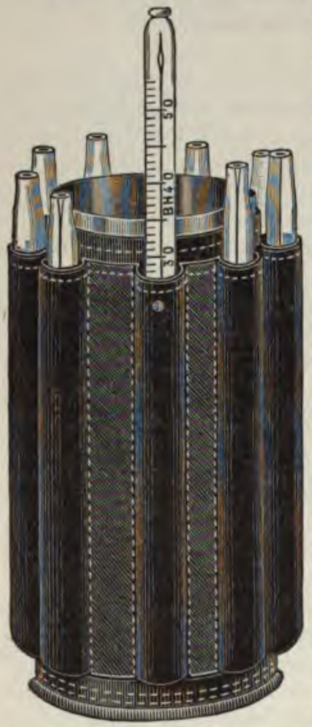

Fig. 23.—Wright's coagulometer, showing the tubes arranged round the water-tin.

Professor Wright's instructions are:—(*a*) That the capillary tubes be of equal calibre, 0·25 mm. being proposed as a standard diameter; (*b*) that in each tube the column of blood be of approximately equal length (5 centimetres); (*c*) that the blood be aspirated for some little distance up the tube to prevent its desiccation at the orifice; (*d*) that before filling them the tubes be warmed to blood heat by pouring water at about the normal blood temperature into the central tin; and (*e*) that this degree of heat be maintained as constant as possible—by further additions of hot water if necessary—until coagulation is complete. This precaution will ensure an optimum temperature, and will render uniform the results obtained during the observation or series of observations. It is desirable to allow about half a minute to elapse between the filling of successive tubes; and in ordinary cases the condition of the blood in the first tube should be tested within three or four minutes from the time of filling. If then, on testing the first tube, the blood be still liquid, a longer time must be allowed before examining the blood in the second tube. If, on the other hand, the blood in the first tube is clotted, the next one should be tested at a somewhat shorter interval after filling. The first traces of clot may be most easily detected by blowing out the contents of a tube upon a piece of white filter-paper. By this method information is obtained as to (*a*) the shortest time which is required for complete clotting in a coagulation tube, and (*b*) the longest time during which blood can remain unclotted in a coagulation tube: the mean between these results will afford a close approximation to the true coagulation time of the blood. Normal blood-clots form in these tubes in from three to five minutes.

It is obvious that, if need be, this method may be employed to investigate the action of various therapeutic agents, such as physiological styptics, on the blood.

After use the tubes are best cleansed by passing a fine brass wire through them, and then washing out with distilled water. By drawing up a little absolute alcohol any remaining water may be got rid of; and in its turn the alcohol may be removed by ether. When the last traces of ether have been volatilised, a process which may be hastened by blowing a current of air through the tubes, these are again ready for use.

Brodie and Russell's method.—In certain respects this method possesses superior advantages, and the necessary apparatus is by no means complicated. One advantage is that a minimal quantity of blood suffices for each estimation, and that the readiness with which the instrument can be cleansed enables a number of such estimations to be carried out in a comparatively short time.

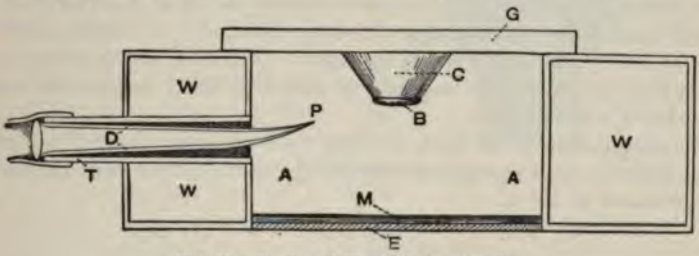

Fig. 24.—Brodie and Russell's coagulometer.

The instrument consists of a deep air-chamber AA, closed below by a glass plate E, upon which lies a layer of water M. It is closed above by a movable glass slide G, to the centre of which is cemented an inverted truncated glass cone C. The whole is surrounded by a water-jacket WW. Inflow and outflow tubes to the water-jacket (not represented in the diagram) enable us to vary the temperature of the air-chamber at will. A metal tube T pierces the water-jacket, and to its interior is fitted a glass tube D which tapers to a fine orifice at P. This orifice lies below the lower surface of the cone C, but is directed towards it.[1]

In using the apparatus the glass plate and cone are removed, and the lower surface of the latter, after thorough cleansing, is dipped carefully into the drop of blood, so that the whole of this surface, but the surface only, is wetted by the blood. This precaution ensures that the drop which is taken up shall always be approximately of the same size. The hanging drop is then brought into the air-chamber, the whole process being carried out as rapidly as possible. The instrument is next placed on the stage of the microscope, and the drop of blood observed under a low power, when, on blowing air gently through the tube D, the contained corpuscles will be set in motion, a weak and short current of air being sufficient for the purpose.

[1] The apparatus is made by Mr. A. E. Dean, jun., 73 Hatton Garden, E.C.

Observation should be confined to the edge of the drop, as the layer of blood here is thinnest, and the view of the corpuscles consequently better than in the deeper layers. It is at the free edge also that clotting is always first noted, the process gradually extending inwards at a rate which depends, to a large extent, on the surrounding temperature.

At times it is found that at the very edge of the drop the corpuscles remain quiescent, but this is exceptional. Ordinarily they move freely right up to the edge of the drop, such movement consisting in changes in their position relatively to one another, as well as in a rotation of the whole mass. On the other hand, as soon as the rim at the edge becomes solid, blowing simply causes an indentation of this rim without causing rotation of the corpuscles.

This stage it is which should be observed, as it is much more difficult to judge with any accuracy the time at which the whole drop becomes coagulated throughout.

In order to obviate any chance of fallacy as far as possible it is well to avoid unnecessary agitation of the drop of blood. We must blow very softly, and only for brief periods of time. In the following tables it will be seen that the method is capable of affording fairly concordant results in a series of observations.

The coagulation times here set out were obtained from the blood of normal individuals, the experiments being carried out in each instance at a temperature of 30° C.

(1) 3·33	(1) 3·10	(1) 3·24
(2) 3·30	(2) 3·00	(2) 3·18
(3) 3·30	(3) 3·50	(3) 3·30

(1) 4·43	(1) 3·00	(1) 3·25
(2) 4·40	(2) 2·55	(2) 3·40
(3) 4·00	(3) 3·50	(3) 3·35
(4) 4·50		

The first two sets of observations were taken on successive days in the same individuals.

VII. Spectroscopic examination of the blood.—It will occasionally happen that information of value from the clinical point of view may be gained by means of a spectroscopic examination of the blood, which could not be easily obtained by any other method. This will be especially the case in poisoning by carbon monoxide and coal gas, or by such substances as amyl nitrite and potassium chlorate. In certain diseases also information may thus be afforded of the approaching onset of coma. I have been able to foretell the probable appearance of this dangerous complication in several cases of uræmia and diabetes on finding evidence of the presence of methæmoglobin in the blood. Methæmoglobin has also been found in severe cases of cholera and leukæmia.

The discovery of hæmatoporphyrin has been recorded in some eventually fatal cases of sulphonal poisoning.

In suspected cases of hæmoglobinuria the diagnosis may be assisted and confirmed by spectroscopic examination of the serum obtained from a blister, which, in the event of the recent occurrence of a paroxysm of this disease, will show the absorption bands characteristic of oxyhæmoglobin due to the destruction of red corpuscles in the general circulation.

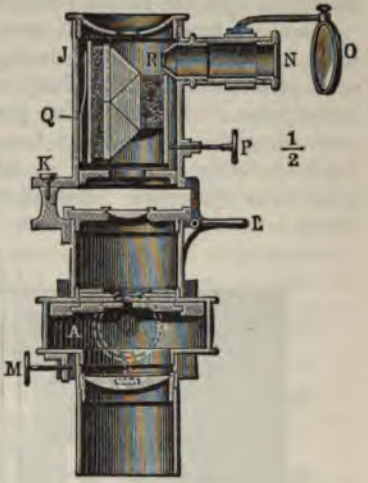

FIG. 25.—Browning's micro-spectroscope.

Method of examination. — The ordinary chemical spectroscope is practically useless in clinical work, the extent of dispersion being too great, and too large an amount of blood being required. These difficulties are obviated by making use of the micro-spectroscope for clinical examinations. Both Zeiss and Browning manufacture small instruments of admirable construction which are employed in connection with the microscope, the micro-spectroscope being substituted for the ordinary eye-piece. The fluid to be examined is placed on the microscope stage, in a tiny test-tube made by sealing one end of a short narrow piece of glass tubing, the cell thus formed being supported on a wooden foot, as at first suggested by Dr. MacMunn. This support serves also to cut off extraneous light. The upper surface of the fluid is now focussed with the ordinary eye-piece, which is then exchanged for the micro-spectroscope. If the amount of material to be examined be extremely small, the high power objective must be employed. In this way I have had no difficulty in obtaining satisfactory absorption spectra from separate crystals of hæmoglobin in a cover-glass specimen of human blood. It is advisable to use artificial light for illumination, as, if daylight be employed, confusion is apt to be caused by the presence of the Fraunhofer lines.

The absorption spectra of hæmoglobin and its derivations.—The normal blood-pigment hæmoglobin is capable of existing in two forms, named oxyhæmoglobin and reduced hæmoglobin respectively, which differ from one another in the amount of oxygen in combination, in the colour of their solutions, and in their absorption spectra. Oxyhæmoglobin, when examined with the spectroscope, shows two absorption bands between the D and E Fraunhofer lines, the intensity of which will depend on the degree of concentration of the pigment and on the thickness of the layer of fluid examined. Under ordinary circumstances these bands are readily visible, even in venous blood which contains a certain proportion of reduced hæmoglobin. The single absorption band, which is characteristic of this variety, and which occupies a position in the spectrum roughly midway between those of oxyhæmoglobin, is somewhat
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diffused and of relatively small intensity. In order to see it clearly it is necessary to treat the blood with some reducing agent such as ammonium sulphide, by the action of which all the oxyhæmoglobin eventually becomes converted into the reduced variety.

A third modification of hæmoglobin which, as previously stated, has been found in the blood during the course of certain diseases and in cases of poisoning, particularly with potassium chlorate, is named methæmoglobin. This contains precisely the same amount of oxygen as oxyhæmoglobin, but differs from the latter in that its reaction is acid while the other two forms of hæmoglobin are alkaline, and also in its absorption spectrum. Instead of the two bands of oxyhæmoglobin it shows four bands, of which one between the C and D Fraunhofer lines is most definite. A very similar four-banded spectrum is also presented by acid hæmatin (or hæmatin in acid solution), but the two may be distinguished by the fact that when methæmoglobin is treated with ammonium sulphide, reduced hæmoglobin is produced, while treatment of acid hæmatin with the same reagent results in the formation of reduced alkaline hæmatin (see Fig. 26). The spectrum of Co-hæmoglobin shows two absorption bands resembling those of oxyhæmoglobin, from which, however, they may be distinguished by their persistence on addition of ammonium sulphide.

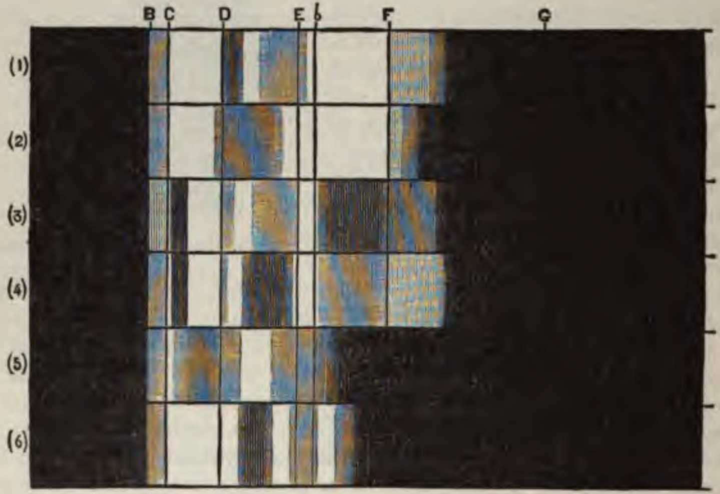

FIG. 26.—Chart of blood spectra. (1) Oxyhæmoglobin; (2) reduced hæmoglobin; (3) methæmoglobin; (4) acid hæmatin; (5) alkaline hæmatin; (6) reduced alkaline hæmatin.

VIII. EXAMINATION OF THE BLOOD FOR PARASITES.—This subject is fully discussed in the articles dealing with the respective diseases in which parasites of one or another kind are met with, but it may be useful briefly to set out certain of the methods employed for their demonstration which experience has shown to be not only efficient but at the same time expeditious. Of the more highly-organised parasites met with in the blood may be mentioned the *filaria sanguinis hominis* and the *plasmodium malariæ*. The ova of the *hæmatobium Bilharzii* have been discovered in large numbers embedded in the walls of the portal vein, but they have not been found free in the blood. Both the *filaria* and the *malarial organism* can perhaps be examined best in the fresh blood, for which purpose a drop of blood should be withdrawn at such time as experience has decided to be likely to give the best results (vol. ii. p. 1065 and p. 724). A perfectly clean cover-glass is then lightly brought in contact with the drop of blood, and afterwards carefully lowered on to a glass slide, so as to produce as thin and even a film as possible. A little vaseline should be smeared round the edges of the cover-glass to prevent evaporation. To stain these organisms, extremely thin films of blood must be taken on cover-glasses and first fixed as rapidly as possible over the vapour of osmic acid, or by dipping the cover-glass in a mixture of equal parts of absolute alcohol and ether. They may then be stained in the eosin and methylene blue mixture originally devised by Canon for the demonstration of the influenza bacillus.

Canon's stain.—Eosin, ¼ or ½ per cent in 70 per cent alcohol, 20 parts; methylene blue, saturated aqueous solution, 40 parts; distilled water, 40 parts. Plehn recommends the addition to the staining mixture of ten to twelve drops of a 20 per cent solution of potassium hydrate. This same method will serve to demonstrate bacteria in the blood. For this purpose, however, it will be necessary, especially if cultivations are also to be made, to obtain the blood with antiseptic precautions; and it is usually better to carry out the preliminary fixing of the film by means of heat, or by treatment with a solution of corrosive sublimate. Staphylococci, streptococci, pneumococci, gonococci, and the bacilli of anthrax, glanders, typhoid, and influenza all take up the methylene blue of Canon's or Loeffler's stains.

Loeffler's alkaline methylene blue stain consists of saturated alcoholic solution of methylene blue, 30 c.c.; caustic potash (1 in 10,000), 100 c.c. When examining for tubercle bacilli it is advisable to make use of Nielsen's carbol-fuchsin solution. The composition of this stain is as follows:—

Nielsen's stain.—Saturated alcoholic solution of fuchsin, 1 part; five per cent solution of phenol in distilled water, 9 parts. The cover-glasses are floated face downwards on this solution, a little of which should previously have been heated in a watch-glass until steam begins to arise from the surface. Two minutes will probably suffice for staining. The superfluous stain is washed off rapidly, and the cover-glass is then placed in a 25 per cent solution of nitric or sulphuric acid in distilled water, until all colour has disappeared. The acid is next removed by thorough washing in water, and the cover-glass is rapidly dried by pressure between two pieces of smooth blotting-paper. If thought desirable, the specimen may be counterstained by a few minutes' treatment with Loeffler's solution.

For the methods of making cultivations a treatise on bacteriology must be consulted.

As a general rule, it is only in the most severe cases that it has been found possible to demonstrate the presence of bacteria in the blood; so that, although such an examination often fails to afford information of any diagnostic value, if positive evidence be obtained the prognosis will be very unfavourable.

This rule has been insisted on by Ely, who has shown that, although the results of bacteriological examinations of the blood have proved disappointing as an aid in diagnosis, yet, by affording an explanation of complications in diseases which are usually localised, they are often of value from a pathological point of view. Thus he found pyogenetic microbes present in the blood during life in cases of pyæmic osteomyelitis, puerperal fever, erysipelas, and infective endocarditis; the pneumococcus in pneumonia and infective endocarditis; the bacillus coli in cystitis complicated with a pyæmic condition; the gonococcus in infective endocarditis after gonorrhœa; the tubercle bacillus in tuberculosis, and the Eberth bacillus in typhoid fever. Block has also put on record a fatal case of typhoid fever in which the bacillus typhosus was twice obtained during life. Kohn has obtained very similar results. He states that in cases of pronounced sepsis large numbers of bacteria may be present in the blood. He also agrees as to the grave prognosis which is indicated by the discovery of the pneumococcus in the blood in pneumonia; a series of negative results, on the other hand, being distinctly favourable to the patient's chances of recovery. Thus of nine cases of pneumonia in which he was able to demonstrate the pneumococcus in the blood, no less than seven were fatal; the other two being complicated with empyema and multiple abscesses respectively. On the other hand, of twenty-three negative cases eighteen recovered, the fatal termination in the other five being due to various complications.

The Widal-Grünbaum method for the diagnosis of typhoid fever.—In this method advantage is taken of the fact that on addition of a small quantity of blood or serum obtained from a patient suffering from typhoid fever to a dilute culture of Eberth's bacillus, loss of motility of the individual microbes is rapidly induced, while at the same time they tend to mass together into clumps, a process to which the term agglutination is now generally applied.

The recent researches of Grünbaum, Wyatt Johnson of Montreal, and of Durham, Wright, and others in this country, have rendered the process at once accurate and simple, and, as it is now possible to obtain the reaction with a single drop of fresh or even dried blood, the method has become readily available in clinical work. Wyatt Johnson's modification of Widal's test is specially applicable to examination of the blood of patients living at a distance. One or more drops of blood are allowed to fall on the surface of a small piece of non-absorbent paper, which when the blood is dry can, if necessary, be sent through the post. To test for the reaction, the drop of dried blood is removed and dissolved up as far as possible in distilled water. This blood should then be mixed with varying proportions of a fresh beef-broth culture of the typhoid bacillus (12-24 hours), or with an emulsion made from a fresh agar culture, in varying proportions, the resulting dilutions of the blood thus prepared ranging from 1 in 15 to 1 in 50. It is advisable that control preparations should be made with normal blood in every instance, and all the specimens should be examined under the microscope within a definite period, preferably half an hour, after preparation.

The serum obtained from a drop or two of blood taken up into a bulbed capillary tube may be examined in the same manner, or it may be added directly to a suitable quantity of a recent broth culture.

Grünbaum's original method is as follows:—A U-shaped capillary tube is filled with a drop of blood from the patient, and the serum separated by centrifugalising. The free extremities of the tube in which the serum collects are broken off, and the serum is mixed with sixteen times the quantity of bouillon. A small quantity of fresh culture of the typhoid bacillus on agar is distributed in 1 c.c. of bouillon, and a drop of the resulting emulsion is added to a drop of the diluted serum. The mixture is then examined as a hanging-drop preparation under the microscope.

It is of importance that the reaction of the culture medium employed for the test growth of Eberth's bacillus should be distinctly acid (preferably 3·5 per cent acid to phenolphthalein in the case of peptone beef bouillon, according to Wyatt Johnson), as otherwise a scum is apt to form at the surface, or a sediment at the bottom of the culture fluid, which in either case not infrequently contains clumps of apparently dead bacilli, the presence of which would tend to obscure the reaction.

At present some doubt exists as to the exact value of the test as an aid to diagnosis, for the reason that it cannot usually be obtained until after the lapse of several days from the beginning of the disease. Again, one or two undoubted cases of typhoid fever have been recorded in which from first to last the reaction could not be obtained. On the other hand, if suitable precautions be taken, it is possible, in the great majority of cases, to demonstrate the reaction towards the end of the first week of the disease, or later. The fact that not infrequently during convalescence the test fails to afford positive evidence in cases in which it has previously been obtainable, tends to show that the specific action of the serum is not dependent on a condition of acquired immunity.

Blood-Crystals.—Under certain circumstances, concerning which as yet comparatively little is known, crystals of one kind or another may form in the blood taken from the body during the course of diseases in which the character and condition of the blood are especially affected.

Among these disorders may be mentioned certain anæmic conditions, more particularly pernicious anæmia, leukæmia, and the various forms in which septic infection may manifest itself.

1. **Hæmoglobin.**—It has long been known that the special blood-pigment hæmoglobin, although of proteid nature, may be obtained with comparative ease in the crystalline form from the blood of some of the lower animals, particularly the guinea-pig and the rat. On the other hand, the hæmoglobin of normal human blood is undoubtedly much more refractory in this respect, since the ordinary laboratory methods entirely fail to bring about crystallisation. Some years ago, however, I made the observation that in specimens of blood from cases of pernicious anæmia, prepared for microscopic examination, rhombic crystals of hæmoglobin not infrequently formed after the lapse of some hours. This was markedly so when the blood had been obtained from persons suffering from a severe form of the disease; especially if a certain amount of pyrexia were present, and provided that treatment with arsenic either had not been begun or had been discontinued for a time. Bond and myself have also noted the appearance of hæmoglobin crystals in blood-films obtained from cases of leukæmia; and the former observer has found that the same phenomenon can be demonstrated in cases of septicæmia and pyæmia. Human hæmoglobin invariably crystallises in the reduced condition, as may be shown by the micro-spectroscope, a point of apparent difference between the blood of man and that of the lower animals. The formation of hæmoglobin crystals in human blood, after removal from the body, is undoubtedly connected with a tendency to abnormal blood-destruction. The readiness or the reverse with which crystals appear in blood-films thus affords some measure of the effect of treatment in restraining such hæmolysis.

The *method* of obtaining crystals of hæmoglobin from the blood in suitable cases is simplicity itself. A fairly large drop of blood, drawn from the finger or elsewhere, is allowed to fall on the centre of a glass slide, and, when sufficient time has elapsed for the edge of the drop to have dried somewhat, a cover-glass is gently lowered upon the surface of the drop. The blood corpuscles gradually break down, and crystals of reduced hæmoglobin will become apparent, in from ten to forty-eight hours, without further preparation.

2. **Hæmatoidin.**—The presence of this substance, a derivative of the blood-pigment, has been detected in an amorphous form by Von Jaksch in the fresh blood of a child suffering from hereditary syphilis. It is frequently found in crystalline form in old cerebral blood-clots, splenic infarctions, and blood-cysts. Occasionally these crystals, or fragments of them, are found within the substance of white corpuscles in the circulating blood under such pathological conditions as obtain in pernicious anæmia and leukæmia, during the course of which diseases minute hæmorrhages in various parts of the body are of not infrequent occurrence.

3. **Charcot-Leyden crystals.**—Occasionally, as in a case recorded by Ord and myself, long colourless pointed crystals have been found in

preparations of leukæmic blood. Their occurrence has not been noted in freshly-drawn blood, but crystals apparently identical with them are not infrequently to be found in the sputum, the fæces, and the seminal fluid. Comparatively little is known as yet of their chemical composition or pathological import.

Certain other methods of blood examination, of which no detailed account will be given, demand brief notice, either because they appear worthy of further investigation, although not yet rendered applicable to clinical needs, or because, although not considered of special value by myself, they nevertheless have been authoritatively recommended or somewhat extensively employed by others.

Under the first category may be mentioned the work of Graham Brown and of Huerthle on the determination of the *viscosity coefficient* of the blood. These observers find that even slight alterations of the "viscosity coefficient" entail an enormous difference in the work thrown upon the heart in propelling the blood round the circulation. Dr. Graham Brown, moreover, has demonstrated the great alteration in the "viscosity coefficient" produced by a change of only a few degrees in the body temperature, the blood flowing much more readily at fever temperature than under normal conditions.

Determination of the *isotonic coefficient* of the red corpuscles is another method of blood examination, concerning the clinical significance of which but little is known. The "isotonic coefficient" is usually measured by the amount of NaCl which it is necessary to add to distilled water to prevent it, when added to blood, from causing its usual destructive effect on the red corpuscles.

The quantity of salt required may, under pathological circumstances, be either greater or less than the normal amount (0·44-0·48 per cent), indicating respectively an increased or diminished power of resistance on the part of the red corpuscles.

The estimation of the number of red corpuscles in a given volume of blood, if it is to be accurate, involves no little expenditure of time and trouble. In the hope of obviating this difficulty, certain *methods of indirect estimation* of the corpuscular richness of the blood have been introduced. Thus, in this country Dr. George Oliver has advocated the use of a hæmocytometer devised by himself, in which the number of corpuscles is gauged by the amount of water which must be added to the blood in order to allow the passage of a ray of light through the mixture. Considerable fallacy, however, is likely to be introduced by any increase or decrease in the volumes of individual corpuscles, or by increase in the number of leucocytes, such as we find, for instance, in leukæmia.

In America the Hedin-Dalland hæmatocrit has been much used for the purpose of estimating the number of red corpuscles in the blood. This instrument is practically a small centrifuge driven by hand with the aid of a system of multiplying wheels. Each of the two arms of the instrument is arranged to hold a small tube of capillary bore, which is marked off into a hundred equal divisions. In use these tubes (or one of them) are filled as accurately as possible with blood and fixed in place in the machine, which is then run for about a couple of minutes. The red corpuscles thus become packed together at one end of the tube. On removing the tube and placing it on a sheet of white paper, it is fairly easy to read off the number of divisions of the scale corresponding to the dense mass of corpuscles. What is really determined by means of this instrument is of course the *volume* of the red corpuscles, from which their number is empirically calculated, each division on the scale of the capillary tube corresponding, in the case of normal blood, to about 100,000 corpuscles. Obviously little or no reliance can be placed on estimations arrived at in this manner in the case of pathological blood; especially when, as in such diseases as pernicious anæmia and leukæmia, there is much variation in the size and shape of the corpuscles.

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CARDIAC PHYSICS

The Cardiac Valves.—1. *Mechanism of the auriculo-ventricular valves.*—At each systole of the ventricles the tongue-shaped valve-flaps pendent from the margin of the auriculo-ventricular orifices are moved together toward those orifices, and meeting together across them block them. By this means the blood in each ventricle is prevented from returning into the auricle, and under the compression of the contracting ventricle is forced to take its way into the great arteries. Were it not for these valves not a drop of the blood would enter the arteries, so long as the pressure in the latter possessed a value near the normal; but for the valves its issue would be far easier back into the cavity of the auricles where the pressures are low. During diastole of the ventricle the flaps of the auriculo-ventricular valves lie in the cavity of the ventricle with their long axes convergent toward the central long axis of the ventricle. Between the valve-flaps and the inner face of the ventricular wall there is always an interval, and therefore always more or less blood (Baumgarten, Krehl). Manometric observations reveal no increase of pressure in the auricle at the moment of closure of the auriculo-ventricular valves. The discharge of its contents by the auricle into the quiescent and already partly filled ventricle somewhat stretches the slack walls of this latter, and, whether by eddy or otherwise, the valve-flaps are raised toward each other and toward the auricular opening. Then, as the contraction of the auricle passes off, the pressure in the now fully-loaded ventricle becomes higher than in the relaxing auricle. The valve-flaps thus swing together into position, and are moved to meet across the auriculo-ventricular orifice, even before the ventricular systole has thoroughly set in. If the arterial openings of the excised heart be blocked, and through the auricles a momentary rush of water under about 12 inches pressure be allowed to play into the auriculo-ventricular orifices, the valve-flaps rise into the orifice, and come together sufficiently firmly to allow of the inversion of the heart without the escape of a drop of its contents.

The valve-flaps would be forced through the orifice back into the auricle were they not tied down to the ventricle by the chordæ tendineæ attached to almost all areas of their under surface. Each valve-flap shares in a pair of papillary muscles; these latter are so placed in regard to the valve-flaps that the resultant of their combined individual directions of pull lies strictly along the long axis of the ventricular chamber, and at right angles to the plane of the auriculo-ventricular orifice itself (Ludwig).

The auricular face of each valve-flap in its position of closure is convex. The thin contiguous edges of the adjacent valve-flaps are bent abruptly downward, side by side, tightly apposed; the tenuous edges of the membranes bear, therefore, no part of the great strain to which the valve elsewhere is subjected; for these edges, projecting into the ventricular cavity, are supported on both sides by the fluid pressure of the blood in the ventricle. That this is the position of these parts of the valve is proved by the following among other considerations: the chordæ tendineæ which are inserted near the free margin of each valve-flap are much shorter than those inserted into the midrib of the flap.

Regarding the use of the papillary muscles, it has been shown (Roy and Adami) that the papillary muscles begin to contract later than does the rest of the ventricle; as the ventricle shortens from base to apex during systole, the papillary muscles, if they are to afford the chordæ tendineæ a suitably placed support, and to prevent retroversion of the valve-flaps into the auricle, must shorten in order to maintain their distance from the auricular orifice.

It has been suggested (Porter) that the auriculo-ventricular valve-flaps and their papillary muscles aid the filling of the auricles with blood. A considerable negative pressure arises in the auricle during the earlier part of the ventricular systole; this seems to occur at the time of contraction of the papillary muscles, and to be due to their drawing down and flattening the valvular curtains which form so large a part of the floor of the auricles. If so, the auricular cavity would be enlarged, and blood drawn into it from the great veins.

It must not be forgotten that an important detail in the mechanism of the closure of the auriculo-ventricular orifices is the circularly arranged muscle surrounding those orifices, as a true sphincter. This sphincter appears to be important, especially for the tricuspid orifice. In the heart of the bird the tricuspid orifice is unprovided with valve-flaps, and its closure is effected wholly by a muscular sphincter.

2. *Mechanism of the semilunar valves.*—So long as the pressure in the ventricle is below the pressure in the great arterial trunk leading from it,

so long will the semilunar valve-flaps meet across the arterial ostium and occlude it. When examined under a pressure approximately that of the aorta, the valve-flaps are seen to lie apposed across the orifice; if one of the flaps be displaced gently towards its attached border, the other two cusps follow it, becoming correspondingly more stretched. The cusps, therefore, in the closed position of the valve mutually support one another. When during the systole of the ventricle the intraventricular pressure becomes higher than the aortic (resp. pulmonary) the valve-flaps yield, are moved apart, and leave between them a triangular opening.

When the valve is open, the position of the cusps is with their free edge convex toward the arterial wall, but the cusp membrane does not lie apposed to or quite close against the wall. In the open position of the valve the arc formed by the curved wall of the sinus of Valsalva may be said to have its chord approximately represented by the free edge of the cusp. The supposition of Brücke that the cusps when the aortic valve is open are pressed back against the aortic wall, so as to block the entrances to the coronary arteries, is completely disproved.

The closure of the valves seems to be brought about in the following way:—During systole the cavity of the ventricle, where it adjoins the aortic opening, is narrowed by the bulging into it of the contracted muscular wall; it forms, in fact, a narrow channel which ends in the direction of the aorta in the triangular cleft between the semilunar cusps in the wide root of the aorta with its triple circumferential bays—the sinuses of Valsalva. At the place where the narrow stream suddenly embouches into the wide aortic channel eddies are formed, curving back behind the valve-cusps, and constantly tending to bring these together. The cusps are, however, kept apart by the pressure of the blood flowing between them; as soon, however, as that flow ceases the cusps rush together, as it were under the force of a spring. Ceradini's account of the eddies which come into play on closing the valves is as follows:—If in a vertical tube containing water, in which visible particles are suspended, a piston at the lower end of the tube be pushed upward, the water in the axis of the tube is seen to move with nearly twice the velocity average for the whole column; along the face of the wall of the tube the water moves so slowly that the piston overtakes the particles suspended in it. As this occurs the particles are seen to be swept from the circumferential zone by a centripetal current conveying them into the axial stream. Along this they rush upward to the free surface of the fluid, where they sweep outward in a centrifugal eddy to reach the wall of the tube again, there later once more to be overtaken by the piston and swept inwards in a centripetal eddy (inversion). If the ascent of the piston be suddenly checked, the above currents in the fluid are modified to the extent that an actual back flow sets in downward along the inner face of the tube. The result is that at the moment the piston stops, the column of water above it is split into two parts—into an axial cylinder moving forwards and a peripheral layer moving backwards, the two being connected above by a centrifugal eddy, below by a centripetal (inversion) eddy. To this latter is due the bringing together into position the cusps closing the aortic opening. The cusps thus brought together are held so by a mechanical force measurable in the left heart by the product of the difference between the aortic and ventricular pressures into the area of the valve-flap, excluding their margins. It is probable that the cusps are partly supported under this strain by the thick bulging myocardium of the ventricular wall on which they may partly rest.

The cardiac sounds.—In 1810 Wollaston showed that skeletal muscle, when it contracts under the will, emits a sound—the muscle-note. The British Association Committee in 1836 declared the first cardiac sound to be the muscle-note of the ventricles, but their observations were not decisive. Ludwig, in 1868, succeeded in proving clearly that when the heart is so placed as to convey by its mass-movement no shock to any vibrator, and at the same time is so inadequately filled as to exclude the possibility of tension of any of its valve-flaps, the first cardiac sound continues to be distinct.

But it has been shown (Wintrich) by means of resonators that the normal heart-sound consists of two notes, the lower of which only can be considered a muscle-tone. The higher is due to the vibration of the auriculo-ventricular cusps and the column of blood they support. This seems clear from the fact that it can be heard if these valve-flaps are suddenly rendered tight in the dissected heart. The first sound of the heart is therefore found to be due to the vibration of (α) the muscular wall of the ventricles, (β) the auriculo-ventricular valves, and (γ) the mass of blood in the ventricles.

The second cardiac sound has been traced to sudden tightening and subsequent vibration of the semilunar valve-flaps. The vibration of the columns of blood in the aorta and pulmonary artery is also partly answerable for the sound. If the root of the aorta and its valve be cut out and tied to the lower end of a vertical tube filled with blood, and the valve be then rendered slack by gently pushing it up from below, and be then suddenly rendered tense by removing the support from under it, a sound is produced. If next the length of the tube and column of blood be doubled, and the experiment repeated, the sound is lowered in pitch although the tightness of the valve-flap is increased. Hence the resonance of the tube and column of blood rather than that of the valve-membrane is the predominant factor in the sound (Talma). But analysis proves the sound to be compounded of a lower note due to the vibration of the column of blood and a higher note due to the vibration of the valve-membrane. The sudden tightening of the valve-flaps and the production of the second sound occur not at the closure of the semilunar valve-flaps, but quickly after.

Of the sounds emitted from the heart the weakest to hear on the surface of the body is that of the right ventricle; the loudest that of the left ventricle. The aortic sound is usually not so loud as that of the pulmonary artery (Vierordt).

Mass movements of the heart.—The diminution in volume undergone by the heart as its ventricles expel their content of blood is accompanied by a change in its form. If the diameters of the heart *in situ* be measured in the opened chest of a supine animal, it is found that during systole the side to side diameter diminishes much—more than the front to back. That is, in systole the heart becomes more or less ellipsoid in cross-section. Probably in the unopened chest and in the erect position its cross-section in diastole as well as in systole is nearly circular. In systole the ventricles are somewhat shortened; but the apex shifts little; it is the base which moves, descending and coming forward towards the apex. This movement of the base is accompanied by a lengthening of the aorta and pulmonary arteries. The latter causes descent of the base of the contracting ventricles, and the descent compensates the shortening of the ventricles, and retains the apex in contact with the chest wall. The cardiac impulse is a protrusion of the chest wall over the surface of the ventricles at the moment just before the expansion of the artery at the wrist. As the ventricles suddenly become hard their long axis becomes more horizontal to the vertical plane of the chest, and is tilted against the resistance of the chest wall. Around the spot where the soft parts of the chest are protruded by the impulse they are found slightly drawn in at the time of each systole. This "negative impulse" is caused by the shrinkage of heart in the air-tight chest as it empties itself, being followed inward by the lungs and to a small extent by the soft parts of the chest wall under the pressure of the atmosphere.

Graphic records of the cardiac impulse can be obtained by one or other of the different forms of cardiographs. Cardiograms, however, in spite of much attention bestowed on their elucidation, still remain unsatisfactory, on account of their variability and the difficulty of disentangling their component factors.

The filling of the heart.—The factors concerned in the filling of the heart are many. The acceleration imparted by the ventricles to the blood, both mediately through the elasticity of the arterial wall and immediately in the heart, gives the momentum of the inflowing blood. Then there is the excess of static pressure in the great veins over that in the diastolic auricle and ventricle. Contributory is the aspiration by the thorax during the act of inspiring, and also the slighter thoracic aspiration produced by the diminution in volume of the heart itself at each systole. The circulatory effect of the rhythmic decrease in intra-thoracic pressure due to these two causes is illustrated by the pulsatile recession of the brain in the cranial fontanelles. Finally the ventricles and the auricles during their relaxation period generate within themselves pressures lower than the pressure in the veins.

Intra-auricular pressures.—The curve of intra-auricular pressure during the cardiac cycle, when its examination is begun at the outset of the auricular systole, shows—(i.) a systolic rise of pressure, which is synchronous with the period of contraction of the auricle; (ii.) a first diastolic fall of short duration corresponding with the relaxation of the auricle and with the earliest part of the systolic rise of intra-ventricular pressure. It is noteworthy that the closure of the auriculo-ventricular valves causes not even a transient elevation of pressure in the auricle. (iii.) The first diastolic rise of pressure is short, and occurs during the early continuance of the ventricular systole. It may be due to the bulging up of the partition between the auricle and ventricle under the high pressure in the latter. It is absent when by vagus inhibition the ventricle is prevented from beating. (iv.) A second diastolic fall occurs while the intra-ventricular pressure is still rising. It lasts longer than the former fall, and is more marked. Its cause may be in the pulling down of the auriculo-ventricular valve-flaps by the contraction of the papillary muscles, which, as Roy and Adami proved, contract somewhat later than the myocardium elsewhere. (v.) A second diastolic rise occurs as a steady increase of pressure, which continues until the beginning of the diastole of the ventricle. (vi.) The third diastolic fall, best marked when the heart is beating slowly, is due probably to a low pressure generated in the common cavity of auricle-ventricle by the suction of the relaxing ventricle. In a particular case the values of the pressures were in the dog's heart systolic rise 9 mm. Hg.: 5, -10, 5, ·5. The flow from the veins into the auricle is intermittent, ceasing during the systolic and the first diastolic rise.

The filling of the ventricle.—As the systole of the ventricle ends and relaxation of its muscle occurs, a negative pressure is generated in the ventricle. Moens supposed that in the latter part of systole the ventricle developed in itself a negative pressure, but his hypothesis is unsupported by subsequent physiological observations. The negative pressure is at first considerable, but this degree of it lasts for a very short time only (Porter), and is over before the auriculo-ventricular valve-flaps can open; it does not, therefore, help directly to fill the heart. There succeeds a longer period of much slighter negative pressure; this assists, the auriculo-ventricular valves being open, to draw blood into the ventricle from the auricle, and into the latter from the veins. Its importance for the filling of the heart is proportional to its duration.

The intra-ventricular pressure.—The rise of pressure in the ventricle which accompanies the systolic contraction of its muscle proceeds gradually though rapidly. It closes the auriculo-ventricular valves almost at once, but for some $\frac{3}{100}$ of a second, though steadily increasing, it cannot burst open the semilunar valves. This is the period of "getting up pressure," the "prosphygmic interval" as Allbutt terms it. The pressure reaches its maximum in about $\frac{8}{100}$ of a second, and for more than the latter half of this interval the semilunar valves have been opened. The pressure continues to rise, therefore, after the opening of those valves has been effected; nor does it recede far from the maximum until the relaxation of the muscle sets in, about $\frac{20}{100}$ of a second after the opening of the valves. The pressure in the ventricle then drops below the pressure in the aorta, and the semilunar valves close. If the pressure in the arterial system is high, the pressure in the ventricle runs a course somewhat different from the above, for instead of reaching its maximum soon after the opening of the semilunar valves it slowly increases throughout the systole, becoming maximal immediately prior to relaxation (Huerthle). In both cases, however, the curve of intra-ventricular pressure is a relatively flat-topped one, showing a "systolic plateau." As Professor Allbutt wisely says, "It is the function of a healthy heart and arteries to promote the maximum of blood displacement with the minimum alteration of pressures." In the systolic plateau two minor undulations of pressure are seen; the causation of these, which are synchronous with two seen in the aortic pressure-pulse, is not clear. On the setting in of relaxation of the ventricle the pressure, in $\frac{4}{100}$ of a second, falls from between 150 and 180 mm. Hg. to below zero; and then for $\frac{4}{100}$ to $\frac{8}{100}$ remains negative. The negative pressure generated varies much in amount, but may reach nearly 20 mm. of Hg. Gradually the pressure rises to a little above zero, and remains a few millimetres above zero throughout the rest of the diastole, until the auricular systole occurs and drives it slightly up to about 10 mm. of Hg.

	Secs.	Secs.
Systole of ventricle before the opening of the semilunar valves, while pressure is still getting up	·03	
Continued contraction of the ventricle and escape of blood into aorta	·27	
Total systole of the ventricle	—	·3
Diastole of both auricle and ventricle, neither contracting passive interval	·4	
Systole of auricle (about or less than)	·1	
Diastole of ventricle, including relaxation and filling, up to the beginning of the ventricular systole	—	·5
Total cardiac cycle		·8

It is important to note that with a frequent pulse the frequency is obtained without appreciable shortening of the cardiac systole, and almost entirely by reduction of the resting period of the heart, the diastole. Further, with a high arterial pressure the period of complete ventricular relaxation is somewhat shortened.

The work of the heart.—The heart is a machine which converts chemical energy into heat, electrical difference, and mechanical work. Only the last-named form of its output of energy need be considered here. During $\frac{1}{3}$ sec. of the ventricular systole the left ventricle exercises a pressure on its contents often amounting to close on 200 mm. Hg.; that is, a pressure of 272 grammes on each square centimetre of its internal surface; 100 cubic centimetres is a low estimate of the output of blood.

By Torricelli's theorem, the velocity v of a fluid streaming through an opening in the floor of a vessel under fluid pressure H is $v = \sqrt{2g\text{H}}$, where g is the acceleration of gravitation (9·8 metres). The kinetic energy E of this fluid is, if m is the mass of the fluid, mgH. And $mg = w$, the weight of the fluid, so that $E = w$H. In other words, a drop of the fluid starting from an orifice in the vessel will have at that orifice the same velocity as if it had fallen freely from the level of the top of the fluid; and the kinetic energy can be measured by the work required to raise it again to a height equal to the height of the top of the fluid in the vessel. If the ventricle be assumed to have no external resistance to overcome in expelling the blood, its work W would be (V being the velocity which the blood would have under a fluid pressure corresponding to the pressure exerted by the ventricle) $\frac{mV^2}{2}$. But the ventricle in expelling its blood has to do so against a high resistance: only a portion of its energy is employed in imparting velocity to the blood. The total pressure is divisible into two parts, I. and II.,—I. spent in overcoming resistance in the tubing of the blood-vessels, II. the flow-producing pressure or velocity pressure. The mean velocity can be ascertained by experiment; its value per second is

$$\frac{\text{Volume of outflow}}{\text{Sectional area} \times \text{time (seconds)}} \text{ or } \frac{Q}{\pi r^2 t}, \text{ and II.} = \frac{V^2}{2g}.$$

The portion of the work of the heart which is used in overcoming the resistance is the difference between the whole work and that quantity arrived at above for II. This latter amounts to about 1·28 grammetres. If the velocity of the blood in the aorta be taken at ·5 metre per second, and the quantity ejected from the ventricle at 100 c.c., and the pressure in the aorta to average 150 mm. Hg., we have the work of the left ventricle amounting to 204 grammetres done against external resistance + 1·28 grammetres represented by the momentum of the moving blood: a total of 205·28 grammetres. The work done by the right ventricle against external resistance may be taken at about 81·6 grammetres. The energy of the muscular contraction directly expended in imparting velocity to the blood is quite small in comparison with the amount expended in distending the arterial wall. The arterial wall, and especially the aorta, is to the heart as a high-pressure cistern to the pumping-station that replenishes it, as its air-chamber to the fire-engine, or as the bag to the bagpipes (p. 476). And probably 100 times more of the heart's work in moving the blood is expended on it indirectly through the aortic arterial cistern than directly on the blood itself. The potential energy entering the heart in chemical form is transmuted to the potential mechanical energy of the heart wall, then to the kinetic energy of accelerated material, and again to the mechanical potential energy of the blood-vessel walls, ultimately to be converted into heat. In the tensions and relaxations of the arterial walls, and in the friction of the moving blood, the heart's energy is continually being converted into heat. In this form the contractions of the heart yield about $\frac{1}{50}$th of the total daily heat production. With a pulse frequency of 72 per minute the work produced by the heart is nearly 25,000 metre kilogrammes in the 24 hours—work more than equal to lifting itself six times in the 24 hours from the sea-level to the summit of Mount Everest.

Experimental observation shows that the heart is a machine which maintains under varying circumstances—so long as its nervous system and its own nutrition are not interfered with—a curiously constant action in two respects; namely, in the duration of the ventricular systole and in the quantity of the output of blood into the aorta. To keep these constant the heart has under varying circumstances to perform very various amounts of work. When the aortic pressure is high, it is found by direct measurement that not only is the maximal pressure produced in the ventricle at each systole much higher than when the aortic pressure is low, but also that the average pressure in the ventricle during systole is much higher than when the heart is beating against a low aortic pressure. The systolic pressure-plateau is much heightened. High arterial blood-pressure involves, therefore, a greater expenditure of energy by the heart at each systole. It is interesting to note that a rise of arterial pressure is in most cases followed by a reduction of the frequency of the heart's rhythm. This is in consequence of excitation of the vagus centre; the stimulation being in part a reflex started from the wall of the heart itself, and in part a direct effect of the high pressure of the blood circulating in the brain. An important factor determining the work of the heart is the distension of the ventricular cavity in diastole. The pressure on a unit of surface of the cavity remaining the same, the total intra-ventricular pressure will vary approximately as the square of the radius of the cavity if the cavity be taken as approximately spherical. Thus Roy and Adami have pointed out that distension of the ventricle means not only increase of the tension of the muscular fibres, but also increase of the lateral pressure on their surface in proportion as the square of their increase in length. But, as they further pointed out, the content of the cavity increases as the cube, and the muscle fibres in order to expel the same constant quantity of blood from the dilated as from the undilated ventricle need to shorten to a relatively less extent than was required of them before. The effect of diastolic distension is therefore, if the output from the ventricle at each systole remain the same, to leave a larger residuum of blood in the ventricle at the end of systole. Recent investigations (Roy and Adami, Huerthle) have shown that to suppose that the ventricle empties itself completely at each systole is erroneous. Not only does it not do so, but the residual quantity of blood varies a good deal, and with it varies generally the amount of distension of the ventricle in diastole. The amount of distension of the ventricle, in other words, the degree of stretch in the muscle-fibres, at the moment when they enter into contraction, is an important determinant of the force of their contraction. All muscles respond by greater contraction when stretched than when unstretched. This increase in contraction is seen chiefly in increase of the work done, and the amount of actual shortening of the muscle is usually less when it is placed under considerable stretch than when it is not. The work done (lift × load) and the heat given out are, however, greater. The ventricle when well loaded, or even excessively loaded, may from our general knowledge of the effect of tension on all muscular structures be expected to expend more energy and do more work at each contraction than when lightly loaded. But it does not necessarily follow that a largely distended ventricle is during diastole more loaded, that is, under higher tension than one only normally distended. The tonus of the heart-muscle is variable, and its tension will depend on the tonus. Moreover, the heart may be considered an after-loaded muscle, its load only coming into play during its contraction. The amount of blood expelled at each systole will be increased in a largely distended ventricle, and is found by experiment to be increased; but at the same time the nervous system is likely to be excited to reduce the frequency of repetition of the heart's beat, and in that way to spare the expenditure of energy by the muscle-cells.

The diastolic size of the ventricle also influences the contraction of the ventricle in another way. The mechanical condition of the contraction of the ventricular muscle differs in one respect remarkably from the conditions obtaining in the skeletal muscles: in the skeletal muscle the contractions are in the execution of most movements approximately isotonic; that is, while the length of the muscle alters, its tension remains approximately constant; broadly taken, it is only in using the muscles for fixation that the contraction becomes isometric, that is, without change in length. The contraction of the heart during the time of getting up pressure is, on the contrary, practically isometric. The muscle-fibres can only alter their length in so far as the cavity of the ventricle can be altered in its shape, its volume remaining constant. But the larger the chamber of the ventricle the smaller the amount of shortening, which, as explained above (Roy and Adami), is necessary for reducing the size of the chamber by a given volume. The output of the heart remains fairly constant for each systole. The amount of systolic shortening of the cardiac fibres then is less when the diastolic ventricle is largely distended than when it is little distended. The contraction in the former case approximates nearer to the isometric condition than in the latter.

In many morbid conditions increased work is thrown upon the heart. In mitral and in aortic regurgitation the ventricle is not an "after-loaded" muscle to the extent it normally should be; its load in those cases is applied in diastole owing to the excessive filling of the heart by backflow. Similar increased diastolic volume of the heart may be brought about by compressing the abdomen and the veins therein (Roy and Adami). As stated above, a certain amount of diastolic loading is favourable to the heart's contraction. In aortic stenosis an extra load is imposed on the ventricle at each systole. The heart is more than normally after-loaded; and here again high-tension of the muscle is, within limits, a favourable condition for output of energy by the heart. But tension beyond a certain degree, and applied for more than a short period, is harmful here as in the case of other muscles. The heart, as Roy and Cohnheim have so well insisted, offers remarkable examples of the reserve power characteristic of the mechanisms of the animal body. By artificially reducing the lumen of the aorta even greatly, the aortic blood-pressure is but little lowered; it is maintained by the expenditure of perhaps a fourfold amount of work by the ventricle, as has been proved by manometric measurements. And furthermore increased activity within limits in the cardiac muscle leads, as in other muscles, to growth and further development of the muscle. To a certain extent, therefore, the heart possesses not merely a great temporary reserve power, but in virtue of its reaction of "hypertrophy" a high degree of permanent reserve power.

The peripheral resistance to the heart.—Our knowledge of the conditions of resistance offered in the circulation of the blood to the action of the heart can be satisfactorily dealt with from a physical point of view only by use of laws which connect together certain measurable facts concerning the blood-vessels and the blood. We require to know the amount of motive force which, as shown above, may be taken to be the aortic blood-pressure, the velocity of flow of blood, and the resistance which is overcome by the streaming blood. The last-named—the resistance—is composed of *two factors, the one resident in the dimensions of the channel, the other in the properties of the fluid—the blood.*

It has just been said that of the factor resident in the properties of the vascular channel the *dimensions* only are of account. The resistance which the channel offers to the flow of fluid along it diminishes with the shortness of the tube and with the increase of the bore of it. The nature of the material composing the tube is practically without influence on the flow. A tube of given dimensions offers the same resistance to a stream of water within it whether it be of metal, of glass, or of any other material. Every moving fluid streams along in a channel lined by its own fluid particles, and the layer of fluid immediately next the wall of the containing channel is practically at rest.

The factor depending on the properties of the blood itself is measurable in terms of standard fluids, and is due to what is called *viscosity*, its internal friction. Fluid flowing along a channel may, of course, be considered as though composed of a number of concentric fluid cylinders ranged round an axial thread of quickest stream, and contained within an outermost sheet where velocity is reduced to zero. In their streaming motion, therefore, the particles of the fluid move over and among their fellows, and this relative movement is opposed in the fluid by its specific coherence or viscosity. As to its degree, this internal resistance is largely influenced in one and the same fluid by temperature. Dr. Graham Brown has proved experimentally that the blood flows with considerably less resistance along tubes when warmed to fever heat than it does at normal body temperature. The internal friction of distilled water is decreased 250 per cent by raising its temperature to blood-heat as compared with its internal friction at 0·5° centigrade.

Between the dimensions of a channel, the pressure-head feeding it with fluid, and the quantity of fluid output from it, certain laws of relation are known. *Poiseuille's "law"* discovers the amount of fluid escaping from any tubular channel of known dimensions fed under a known pressure-head. The output Q in unit time varies, when the length of the tube is very great in comparison with its diameter, directly with the fourth power of the diameter d of the tube, and with the feed-pressure h; and inversely with the length l of the tube.

$$Q=\frac{d^4h}{l}.$$

From this, by comparing outputs of various fluids with output of a standard fluid (distilled water), the coefficient of internal friction c can be obtained; so that

$$Q=c\frac{d^4h}{l}.$$

Poiseuille's law is in all hydrodynamics perhaps the nearest approach to accord between theory and practice. It is, however, formulated for conditions which are not very approximately those existing in the circulation of the blood. It deals with flow along cylindrical tubes under constant pressure. The blood-vessels are but approximately cylindrical, and we shall probably never be able to ascertain their particular dimensions from moment to moment, as, under the influence of the nervous system, we know they must be continually changing. Again, they are curved and complexly branched; furthermore, the pressure in the blood-channels is to a large extent a pulsatile one. The last difficulty has been especially investigated by Huerthle. He finds that, in experiments carried out with distilled water, a *pulsatile pressure* resembling that in the arteries did not seriously upset the Poiseuille law so long as it could be accurately averaged. The results obtained under the pulsatile pressure harmonised well with results obtained under an equal average but constant pressure. A further difficulty lies in the blood being not purely fluid, but a fluid containing semi-solid bodies suspended in it. The inspissation of the blood, which constantly occurs as an element of "collapse," in acute cholera, and so forth, probably alters greatly the viscosity coefficient of the circulating blood.

These circumstances complicate the application of Poiseuille's law to physiology. It is, however, possible for a given brief period to ascertain certain data which are of use in forming a conception of the amount of physical resistance which the heart overcomes in maintaining the circulation. We can determine the fluid pressure in a vessel, the amount of blood flowing through that vessel, and the viscosity coefficient of the blood of the animal at the time and at its normal temperature, also in some cases the diameter and length of the channel. Huerthle has recently determined *the viscosity coefficient.* He allowed the blood to flow direct from the carotid through a calibrated tube, and measured contemporaneously the outflow from the tube and the pressure-head feeding the tube with blood. The blood was successfully prevented from cooling, and all fear of interference from clotting was avoided by reducing the period of flow to less than thirty seconds. The time was measured in hundredths of a second, the quantity of outflow in cubic millimetres. In this way the following results were arrived at:—

(i.) The coefficient of viscosity of the blood is in one and the same species of animal relatively constant.

(ii.) In one and the same individual animal the viscosity of the blood when measured by observations with tubes of various size and with various heights of arterial pressure is found to give an almost exactly identical coefficient. From this it would seem that the suspension of the corpuscles in the blood does not seriously affect the application of Poiseuille's law to it as a fluid.

(iii.) The average coefficients of viscosity for the blood of different species examined were found to be, compared with water at 37° C. as 1, as follows:—Blood of dog, 4·5; of cat, 4·1; of rabbit, 3·2. Of these, that of the dog probably most closely approaches that of man.

With the data of pressure-head, quantity of outflow, and viscosity coefficient found, there remain in Poiseuille's formula only two unknown quantities, namely, (d) *the diameter* and (l) *the length* of the *cylindrical tube-channel*. If d be represented by a definite number, as in certain cases it can be, then we can solve the equation, in so far that we can determine the length of a tube through which the same quantity of blood would flow in unit time—as through the aorta—and in that way obtain a definite expression of the amount of resistance in the circulation along the aorta.

Huerthle finds in this way that *the aortic channel* of the rabbit offers to the blood-flow a resistance equal to that which would be offered it by a cylindrical tube of the same diameter as the aorta and 300 m. in length. Again, making use of data h, the arterial pressure, and Q the quantity of outflow of blood from *the renal artery*, and d the diameter of the renal artery under a pressure of 100 mm. Hg., Huerthle found the resistance offered in the vascular path through one kidney. The resistance offered by one kidney (dog) weighing 100 gram. is equal to that which would be offered by a tube of the same diameter as the renal artery (of the dog), namely, 4·6 mm., and having a length of 35 metres. Under the influence of diuretics this resistance becomes greatly lessened, so as to correspond with a tube of similar diameter, but only 22 metres in length.

One very remarkable conclusion from the above is that the resistances offered in the aortic channel and in the renal respectively are so greatly different as probably to indicate a profound difference in function. In comparing the resistances offered in the two channels, we can express both in tube-lengths of 30 m.; but then the diameter of the aorta must be reduced in the tube that represents it to 4·5 mm. That is, in other words, *the aorta has a relatively greater diameter than the renal artery.* This leads to the conclusion long ago introduced by E. H. Weber, that the aorta, with its peculiarly elastic wall, is not merely a channel, but a distensible reservoir for the blood thrown out by the heart; it stores up this blood temporarily for distribution during diastole (p. 471).

In using Poiseuille's law it must be remembered that it cannot be applied directly to solve the relation between speed of blood-stream and height of blood-pressure in the animal body, although this is sometimes done. Inasmuch as the vascular channels are extensible, and their diameter therefore variable under alteration of blood-pressure, the relationships that hold good between pressure and velocity in rigid tubes will only obtain in modified degrees. The innervation remaining unaltered, we may assume that to raise the pressure twofold in a distensible tube will more than double the velocity.

But the main portion of the work of the heart is expended immediately, not on moving the blood through the vessels, but in *stretching the arterial wall*. The elasticity of this wall is therefore of importance in physical action of the heart. Wertheim and Roy examined the elasticity of the aorta by hanging weights on a strip of uniform cross-sectional area taken from it. Roy, by an ingenious apparatus, obtained continuous graphic records while the load was uniformly increased in weight, and thus obtained curves in which the weights are represented by the abscissæ, the elongations constituting the ordinates. Both he and Wertheim agree that the curve obtained (if the strip be fresh and from a healthy vessel) is an hyperbola. Roy and Zwaardemaker have further examined experimentally the increment of cubic content of the vessel obtained under heightened pressure. Starting from a pressure about equal to that normal in the blood-vessel under examination, they found that, under successive equal increments of pressure, the increase obtained in capacity is greatest at first; and as the pressure is gradually heightened, the increase in capacity obtained becomes less and less. They found also that as the pressure starting from normal (for example, 120 mm. of mercury for the carotid of the cat) is reduced by successive equal decrements, the diminution of capacity follows more rapidly at first than later. These observers, therefore, find the extensibility at its greatest at a range of pressures which are frequent and usual in the vessel under examination. Also that above those pressures the curve of extensibility is hyperbolic. It is clear, therefore, that with a high arterial blood-pressure a certain further absolute increase of pressure will distend the vessel less than will the same absolute increase of pressure under a lower arterial pressure. Also that the injection by the heart into the aorta of a certain absolute quantity of blood will raise the arterial tension relatively more when the pressure is already high than when it is about the mean or is low. The walls of the smaller vessels have been proved to be more easily distensible than those of the large, so that any increase in the amount of blood in the arterial system will locally distribute that blood in the smaller or larger vessels relatively differently under low than under high arterial pressures.

It is noteworthy that the rupturing strains of the arteries is proved by experiment to be about twenty times greater than any strain the body can put upon them; this is true, of course, of healthy vessels.

Influence of the force of gravity on the heart.—It might at first sight appear that since the blood in circulation lies practically in a vertical circuit, the effect of gravity as regards the work to be done by the heart in maintaining the movement of the blood would not be affected by gravity, the weights of the blood in the up-stream and down-stream columns balancing one another. But such a view leaves out of consideration the effect of the static pressure of the fluid columns in the vessels in stretching the walls of the vessels. L. Hill has recently investigated the results of this for the heart and the circulation generally. In respect of the former he points out that the force of gravity must be regarded as a cardinal factor in circulatory problems. The splanchnic vaso-motor system is entrusted with the important duty of compensating the hydrostatic effects of gravity brought about by changes in the posture of the body. This action of the splanchnic vaso-motor system is far more developed in upright animals, such as the monkey, than in rabbits and dogs; and therefore is probably very complete in man. He proves that when the power of compensation is damaged by paralysis of the splanchnic vaso-constrictors, for instance by shock, in asphyxia, or by chloroform, the blood drains into the abdominal veins, the tonus of the splanchnic vessels not being sufficient to resist the hydrostatic pressure if the upright position be assumed; in consequence the heart empties and the cerebral circulation ceases. In the horizontal and in the "feet-up" position syncope is avoided or recovered from, the force of gravity acting in the same sense as the heart. To bandage the abdomen firmly has the same restorative effect. Chloroform by destroying the compensation for gravity in the circulation can kill an animal if the posture be one in which the abdomen is on a lower level than the heart.

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CHLOROSIS

Syn.—Latin, *Morbus virgineus* (Lange, A.D. 1520); German, *Bleichsucht*; French, *Pales couleurs*; English, *Green-sickness.*

[Professor Stockman tells the author that the name Chlorosis was given to this disease by Jean Vavandal in A.D. 1620.]

Introductory.—Anæmia.—That in the course of many diseases the blood should vary in composition, chiefly in the direction of impoverishment, is to be expected. It may thus vary in more than one quality; it may vary in mass; in plasmatic value; and in corpuscular value. In pining, for example, we note loss of water, loss of plasma, and loss of red corpuscles; as proteids fail, the water, which is retained more or less loosely by them, tends to escape; finally, the corpuscles lose their vigour and the activity of their growth. We have no means of measuring the fluctuations of the mass of the blood with any approach to accuracy; still it seems certain that the blood does vary in mass; sometimes in the direction of excess, more frequently in that of defect. Smallness of arterial pulse is no guide in this matter; the artery under observation may contract upon its contents so as to produce a relative anæmia of a particular area; or a general arterial anæmia may coexist with a venous plethora, the mass of the blood not being diminished. It would seem, however, that in some diseases, such as cancer or exhausting discharges, and in old age, the mass of the blood is diminished; the arteries are unfilled, and there may be no sign of venous distension in any area. It seems probable also that in the anæmia of young men the mass of the blood decreases; in chlorosis it does not fall, and is supposed even to rise (Rubenstein and James).

Of the variations in the composition of the plasma and in corpusculation we have better evidence as, by methods described in a previous article (p. 408), we are enabled to submit these constituents to direct estimation. In the present article we have no concern with excessive values, our text is poverty of the blood. Moreover, seeing that anæmia is a factor in many diseases, I must refer the reader to other articles of this work—as to those on pernicious anæmia, splenic anæmia, leukæmia, wherein the blood changes are eminent; or to phthisis pulmonalis, chronic dyspepsia or diarrhœa, wherein the anæmia is rather a secondary event—in which certain deteriorations of the nutritive fluid are particularly described. We have some concern here, however, with anæmia occurring in the course of temporary deviations from health as distinguished from that of maladies in which the defects of the blood are of a secondary kind. Apart from the graver maladies we are all of us familiar with states of debility and lack of colour and condition which, at whatsoever time of life they may come on, we attribute, and often with reason, to a temporary and curable impoverishment of the blood. There are certain times of life when we may be too ready to put down any such flagging of sanguification to transient causes, as for instance in boys and girls in whom the demands of growth and development are extraordinary; there are other times of life, as for instance in advancing years, when we are on the alert to see in the change a herald of organic disease, and may be happily deceived nevertheless, for old persons too are occasionally prone to fail in the common task of keeping the blood up to its proper standard; though in them this failure is always of more serious meaning than it is in the young. Again, the blood may be sufficient in mass, and yet deficient either in nutritive value, or in oxidising power; or indeed in both these qualities together. For these various states sundry and somewhat uncouth names have been provided, such as hypalbuminosis, oligocythæmia, and so on; while defect in the mass of the blood has been named oligæmia—names not without their convenience. We also hear of hydræmia as a name for a state of the blood in which the fluid is said to be unduly diluted with water, and thus, if not diminished in actual bulk, defective in proteid matter. It is said that the blood of anæmic young men is not deficient in hæmoglobin, and that they are not very pallid; that it is the quantity of arterial blood, or at any rate of the plasma, in the vessels which is under the standard: the blood does not spring from the finger when pricked as it does in the chlorotic girl. In young men's anæmia, therefore, the specific gravity of the blood may actually rise above the normal mean (Lloyd Jones). Again, the blood may be defective, or, on the other hand, unduly abundant in salts. These variations are less important, as they are probably integral parts of the former changes: for instance, the salts probably depend directly upon the quantities of the albuminous elements of the blood; the water likewise may rise and fall in part with the albuminous elements with which it is more or less loosely combined: moreover, it is dependent upon the saline density of the blood. Intimately speaking, therefore, while the causes of anæmia may be infinite, the number of anæmic permutations may be few (19). As I have said, however, we have in this chapter to deal with anæmia in its dynamical rather than in its statical aspects; with anæmia which, under favourable conditions, admits of more or less rapid readjustment with recovery of equilibrium.

Anæmias of such kinds may be divided into (*a*) those in which consumption of the blood is accelerated; (*b*) those in which renewal of the blood is slow; and (*c*) those in which both sources of failure are combined. Of the first, fever may be taken as an instance; of the second, inanition from whatever cause; and in pulmonary phthisis, if both appetite and digestion be poor, we shall recognise the mode in which undue rapidity of consumption may conspire with imperfect renewal.

It need not be said at large that such conditions as these merge by insensible gradations into health. For example, in growing youth rapid use of the blood may not be made up even by good appetite and digestion; in old age, although the use of the blood may be slow, appetite and assimilation may be slower still. Again, in direct loss of blood, or in the infection of the blood by some poison, recovery of health is to be anticipated. We shall not forget, however, that waste of blood is far more mischievous and dangerous in old persons; and in those, whether old or young, in whom restoration of the plasma and of cell growth is for some reason imperfect.

Apart from the graver diseases, then, we should expect to find anæmia more frequent and more obstinate in the young on the one hand, and in the old on the other. Some persons are anæmic, or have a bent to anæmia, all their lives long; but simple anæmia is less apt to occur in the decades between thirty and fifty, for at these ages perturbations of nutrition are better resisted. Some persons seem to have a richer blood store than others, to resist the incursions of injurious agents more successfully, and to recover more quickly from such incursions.

Symptoms.—Of the symptoms of anæmia I shall say almost enough under the head of chlorosis, to which these considerations are but introductory; still the symptoms of chlorosis are not merely those of anæmia; and it may be well to ascertain how far the phenomena of chlorosis are peculiar to this state, and how far they are common to anæmia of whatever origin. The main distinction in the phenomena is in the condition of the pulse, which in anæmias of failing quantity is not only quickened but also feeble and empty. In chlorosis, as we shall see, the pulse may be full and of good or even of excessive pressure. Another side of this peculiarity is seen in the action of the heart, which in anæmias secondary to serious disease may be feeble and almost impalpable, while in chlorosis it is often irritable and sometimes obtrusive. Fatty changes in the heart are common in their degrees to all anæmias. In chlorosis the anæmia tells rather on the respiration and on the steadiness of the heart; in other anæmias, or in many of them, the effect is rather marked by slackness of the cerebral circulation and syncope. Wasting, generally speaking, is not a very prominent symptom in anæmias, and it is rarely seen in uncomplicated chlorosis. The pathology of anæmia will be discussed incidentally under the chlorosis.

The diagnosis, prognosis, and treatment of anæmia depend upon the primary malady of which the anæmia is a symptom—as of syphilis, plumbism, malaria, and so forth. For the most part anæmia is a symptom rather than a disease; even in chlorosis there is no doubt some specific series of antecedents which as yet is hidden from us. On the other hand, as I have already said, in some persons, in whom the hæmopoietic capacity is habitually low, anæmia may be the primary factor; in these cases the proteid elements of the fluid seem to be as much in defect as the hæmoglobin, yet iron is nevertheless an important means of cure or relief. Thus it would appear that iron, the specific action of which in anæmia is hard to explain, does more than feed the red corpuscle; it seems to possess some property of stimulating the growth of the blood as a tissue.

CHLOROSIS.—**Definition.**—Chlorosis is a malady of women, and primarily of young women at or about the age of puberty; it consists in defect of the red corpuscles of the blood, a defect partly of numbers, chiefly of hæmoglobin; the plasma being constant, or even enriched.

Under one name or another chlorosis has attracted attention from early times, yet it was not until the clinical studies of Hoffmann and of Johann Duncan gave accuracy to the description of the malady that it took a definite place in nosology. Ashwell was the first physician to recognise chlorosis as something more than a symptomatic anæmia; and Hayem the first to place the disease on a firm pathological basis.

Causation.—It would serve little good purpose to dwell on the fanciful views of the causes and characters of chlorosis which have prevailed among physicians and poets—views which are adumbrated by the use of such names as *febris amatoria*, *icterus amantium*, and so forth. We shall see hereafter that the attribution of chlorosis to perverted or thwarted sexual impulses is mistaken, except in so far as an emotional disturbance of whatever origin may contribute to the causation of the malady. On the other hand, although we may have cleared our minds of certain false preconceptions, we cannot yet pretend to be in possession of much more accurate knowledge of the causes of chlorosis. Many and various are the surmises; and of these, or of some of them, I will try to give an account.

Heredity.—That chlorosis is hereditary in no small measure seems to be believed by most observers of the disease, and certainly accords with my own experience. In family after family do I remember the daughters, one after another, as they arrived at puberty, coming for aid in this disorder. It may be replied that as chlorosis is so common a malady it will naturally appear in most or all families as the girls grow up. Still, making all allowance for this confusion and for similarity of conditions, I agree with those who say that chlorosis in its more strongly marked forms tells especially upon certain families; and that in such families the girls are hit more hardly and resist treatment more obstinately than in others. Whether the bent to the disorder may run in a latent channel through the fathers, I cannot say; it seems rather to run through the mothers, as I have found that in families of chlorotic girls the mother commonly says that she and her sisters suffered likewise in a notable degree. I regret to say that I am old enough now to see in my consulting-room the chlorotic daughters of women whom years ago I had treated before their marriage for the same disorder. Dr. Lloyd Jones has published certain opinions on the heredity of chlorosis which I shall more conveniently discuss in the following paragraph:—

Sex.—Between the extreme opinions of Dr. Lloyd Jones and those of Dr. Simon, the one holding that chlorosis is wholly and peculiarly a disease of women, and the other that chlorosis is little more than an anæmia of ill-thriven young people, there is a great interval. Dr. Jones, in a series of papers which are remarkable not only for speculative ability, but also for industrious investigation of the phenomena of chlorosis, expresses some such views as the following:—In chlorotic women the specific gravity of the blood falls: on further inquiry it seems that this fall is due to defect in the corpuscular element, and that when the plasma is tested separately the specific gravity is not only up to the normal standard, but may exceed it. In this important respect the blood of chlorosis differs from that found in ordinary anæmia, in which the specific gravity of the blood tends to rise while that of the plasma alone tends to fall. Again, the serum in ordinary anæmia is deficient in quantity, but that in chlorosis is abundant. The first kind of anæmia (oligæmia) may occur in either sex indifferently; the second is peculiar to women in the child-bearing period. In the treatment of this second kind iron is of specific value, in that of the first its value is less certain.

Dr. Jones then goes on to say that the anæmia marked by abundant plasma and deficient hæmoglobin—that of chlorosis—is peculiar to women, and is found in women who come of large families,—in women who have many brothers and sisters. Since these observations were published I have questioned my own experience, and, so far as impressions go, I am disposed to think that the author is right in this respect. Dr. Jones goes one step farther, and asserts that in large families the blood of the sons as well as of the daughters has the chlorotic bent; its plasma is abundant and of good specific gravity. From these facts he infers that this kind of blood is the blood of fertility; and that chlorosis is the exaggeration of the fertile blood, of blood, that is, which has for its end the storage of nutritive material for the fœtus during pregnancy. That such a leaning should be seen in the blood of women at puberty thus becomes comprehensible. These opinions are based on a large number of observations both clinical and pathological, they are coherent and interesting; whether they are true cannot be settled at present. Meanwhile they hold the field, and they make a good working hypothesis, one which has this in its favour, that, to close observers, perhaps every girl passes, as it were, through the outer court of chlorosis in her progress from youth to maturity. One other point seems to me to be in its favour, namely, that the causation of chlorosis is probably simple; the symptoms being uniform, and general in their incidence on one sex, it is probably due to some widely acting antecedents of a kind not subject to much perturbation. Whether Dr. Jones' hypothesis be true or not, we are probably near the discovery of some such cause of general operation deflected but little by contingent causes. For this reason I think that the conceptions of causes of more multiform or incidental activity, which we shall presently consider, are less likely to be true. Some of Dr. Jones' results, such as the maintenance of the volume of the plasma, are corroborated by Rubenstein; and the persistence of its specific gravity is verified by Hammerschlag. Dr. C. F. Martin says, on the other hand, that if a relative fall in hæmoglobin be taken as a test, chlorosis occurs in men also. He gives four cases, estimated by Fleischl's method (duly controlled), in which with corpuscles from 4,800,000 to 5,300,000 the hæmoglobin fell to 68, 72, 77, 77 respectively; he does not state whether these men were members of large families. My own experience is that the occurrence of chlorosis in men is either unknown or very rare; certainly no observations to the contrary can be accepted unless a careful examination of the blood be recorded.

Race and climate.—We are told that chlorosis obeys no climate, no latitude, no altitude. Hirsch tells us that it is found in Asia Minor, in Algeria, in the West Indies (Creoles), and so forth. I have seen it abundantly in South European races, such as the Italian, and in women of all builds and of all breeding; at the same time, without records of examination of the blood, statements of this kind have but an approximate value. It is said that anæmia is commonest in blondes; and Lloyd Jones adds that blondes are more fertile. I am not satisfied that chlorosis is commoner in blondes; the assertion is open to the criticism that in the blonde it is more conspicuous; indeed the district with which the observer is conversant is no small element in his experience.

Age.—Chlorosis is a malady of puberty; if it occur in later life, as no doubt it often does, the attack may assuredly be regarded as a relapse. All authors agree that a first attack rarely appears after the age of 24. Professor Stockman gives 23 as the highest age of his series. Now in this respect it is remarkable that Leichtenstern found in the age period from 18 to 25 that the hæmoglobin is ordinarily about 8 per cent less than in the period from 25 to 45 years of age. Sörensen substantially corroborates this statement. Stockman (54), in a series of 63 cases, found that no fewer than 41 lay between the ages of 15 and 20. Sörensen attributes this diminution of red corpuscles directly to menstruation; Stockman to the demands of puberty in a more general sense, digestion and appetite being, moreover, often impaired at a time when menstruation is being established.

Conditions of life.—Almost every defect in the circumstances of life has been regarded as a direct cause of chlorosis; that such defects continually intensify the disease is admitted by all observers. To work in a badly ventilated room will keep up chlorosis, or anæmia at any rate, in spite of remedies. Some overwrought and underfed women only keep going by taking iron, from time to time, for the best part of a lifetime. Still, an accelerating cause is not necessarily a principal cause. Mental strain, again, is rather a favouring condition than a direct cause. Dyspepsia, with consequent inanition, takes an important place in the causation. In about one-half of Professor Stockman's cases disorders of digestion were present; so that if the primary cause do not lie in the stomach it is probable that malassimilation is a favouring condition. Dr. Simon lays great stress on the dyspeptic element in chlorosis; he tells us how capricious the appetite becomes in young girls, nay even depraved; such stuff as slate pencil and the like being devoured. There is, indeed, an especial proneness in chlorosis to atonic and perverted gastric functions, if not to actual dilatation of the stomach. Many young women, as their frames develop, fall into a panic fear of obesity, and not only cut down their food, but swallow vinegar and other alleged antidotes to fatness. Nearly all chlorotic girls are disposed to shirk meat and to feed rather on pastry and sweetmeats; and of the meat which is eaten, browned and burnt fragments form no inconsiderable part. If these ingesta do no direct harm, at any rate they conceal a process of inanition; and a fall in nutritious food quickly leads to a fall in red corpuscles. These losses the full-grown woman may recover from readily; the growing and developing girl cannot so easily make up the larger arrears. Yet, after all, as careful observers like Professor Stockman record that only about 50 per cent of chlorotic women are dyspeptic, we cannot regard dyspepsia as a necessary antecedent; the primary cause lies deeper. We have only to look at the peasant girls who come with chlorosis to our rural hospitals, and again at the young maid-servants in good families, to see at once that chlorosis is in its essence independent of food caprices, city life, hard conditions, and indigestion. The healthy country girls may show the malady less; it may fall on them with less average severity the better their conditions of life; at any rate, they may recover more quickly; still chlorosis does not pass them by. Niemeyer testifies to the numbers of robust peasant girls from the surrounding villages who were wont to present themselves before him with chlorosis. Meinert presses this kind of explanation in a special form: he attributes chlorosis to tight lacing or to the belts worn by women; these practices, as he alleges, lead in a considerable percentage of women to splanchnoptosis; he records gastroptosis in most of his patients who suffered from the malady, and in 15 per cent he reported movable kidney. Surely, of movable kidney, at any rate, this is an exaggerated proportion, and one opposed to the reckonings of all physicians who have studied these dislocations (vol. iv. p. 342). As regards the stomach, I feel impelled to surmise that in many cases Meinert must have taken toneless and inflated stomachs for dislocated stomachs; no reports on this subject are worth much unless the line of the smaller curvature be plotted out.

Estimates of the hydrochloric acid present in the stomach in cases of chlorosis have been made by many investigators. In some this acid was found in excess, in some in defect; in others, again, it proved to be normal in amount. Similar results would probably be obtained in any group of sickly young persons.

It is a common experience that many girls otherwise healthy and living under the best conditions of life become chlorotic: perhaps no girl escapes it altogether; some, however, show it but little, and recover rapidly. The secret does not lie in inanition or dyspepsia.

A more potent cause, perhaps, is emotion; either emotion of a wearing and long-continued kind—such as love sickness, home sickness, and the like, or shocks of a more sudden onset. A remarkable case of this kind came under my notice a few years ago. A young lady became very chlorotic, and her cure was not so easy as usual; however, after a little patience she was apparently cured, and the treatment was continued until fear of relapse had abated. On a certain evening, soon afterwards, the other members of her family having gone out till a late hour, she went to bed alone. At midnight she was awakened by a sense of some presence in the room, and on opening her eyes she saw a figure in white moving across it. She lay speechless with terror until the apparition, after some pacings, passed out of the room again. As it issued from the room she became aware that the ghost was the butler in his night-shirt; and she sprang out of bed to bolt the door after him. As she did so he returned towards the door, and, thrusting against it, tried to re-enter the room. With strength renewed by fear she thrust against him, and after some effort she secured the door. The man hung about the landing for some time, and she sat on her bed in an agony of apprehension until her parents' return home, about four o'clock in the morning. It turned out afterwards that the poor man was a sleep-walker, and his promenades innocent enough. On the next day, however, the chlorosis was profound, and she was brought to see me again in a worse plight than before. I was assured by the girl's mother that when they left the patient on the evening of the alarm she was to all appearance well; by daylight next morning she was seen to be as I saw her. Other cases of the same kind are on record. Still, such a mode of causation is uncommon, and probably depends upon a strong proclivity to the disease.

Generative organs.—The fashion of attributing chlorosis to sexual disturbances of which the patient may or may not be conscious is passing away. The final extinction of this hypothesis we owe to Rokitansky and Virchow, who proved by necropsies that no constant morbid condition of the organs of generation is found in these cases: the parts may be normal; or this or that abnormality, such as hypoplasia, may be discovered: but all or any are of an incidental kind, and present no common factor. At the same time, if epithelial débris be found repeatedly in the urine, masturbation must not be forgotten, and corroborative evidence of the habit may be detected.

Mesoblastic hypoplasia.—Morgagni, Meckel, Rokitansky, and, still more definitely, Virchow have drawn attention to a peculiar arrest of development of the arterial system found after death in certain cases of chlorosis. [*Vide* art. "Diseases of Arteries."] Such necropsies are few, for it is only by accident that cases of chlorosis come to the post-mortem table. In the cases before us a very strange state of things is revealed. The aorta may scarcely admit the little finger, and the abdominal portion of the vessel may be no bigger than the ordinary iliac or femoral artery. This remarkable arrest of development is seen to be but a part of a general arrest throughout the whole arterial system, and is supposed to indicate a like hypoplasia of the mesoblastic layer throughout, including the blood-forming organs; hence, it is said, the peculiar anæmia. This explanation is rather of the dead-house than of the bedside. That a disorder so common and for the most part so curable should depend upon a malformation so grave and so incurable as this aortic and general vascular hypoplasia is on the face of it highly improbable. Again, so far as our evidence goes, the arrest may occur in either sex indifferently (Hayem). It is said, indeed, that Virchow was not always careful to exclude the cases of congenital or slow heart disease with which a hypoplasia of this kind may be bound up (Pye Smith). Be this as it may, Virchow's doctrine has a great vogue in Germany; and it would ill become us to deal lightly with a well-considered opinion expressed by a pathologist so eminent.

Hæmorrhage.—Loss of blood is a common and direct cause of anæmia, and has been assumed to be the primary cause of chlorosis. It is said that in many cases hæmorrhage is or has been obvious enough; whether in the form of menorrhagia, epistaxis, hæmorrhoids, hæmatemesis, or otherwise. And it is urged, if hæmorrhage be a *vera causa*, and in a considerable number of cases an immediate factor, may not hæmorrhage be the general cause; hæmorrhage which, if it issue by some passage unobserved, or in repeated quantities too minute to catch the eye, may often be overlooked? Such imperceptible oozings have been supposed to occur into the stomach, for example.

Now we have seen that the anæmia which results directly from hæmorrhage is not quite identical with that of chlorosis; that it is revealed rather by a diminution in the number of the red corpuscles than by their defect in hæmoglobin; this proposition, however, is far from established. Yet there can be no doubt that chlorosis occurs daily in which, after the closest inquiry, no hæmorrhage can be seen or heard of: and in respect of the alleged persistent oozing of blood from mucous surfaces the contents of the stomach have been repeatedly tested without the discovery of any reactions due to blood or sanguineous effusion. That menstruation or other blood loss, even if moderate, may aggravate chlorosis is certain, and amenorrhœa, therefore, is often a protective condition; but on the other hand chlorosis, as we all know, may occur in girls before the appearance of menstruation. The effects of hæmorrhage on the specific gravity of the blood plasms have yet to be determined.

Bunge's hypothesis.—A very ingenious hypothesis in explanation of chlorosis, and of the behaviour of iron in the cure of it, has been proposed by Bunge. I will set forth the hypothesis in the lucid words of Professor Stockman:—

"Bunge holds that the ordinary preparations of iron, including the so-called albuminates and peptonates, cannot be absorbed from the alimentary canal. He points out that ordinarily the iron of the red corpuscles is formed from the organically combined iron in the food, which is something like hæmoglobin in constitution, and can be readily absorbed and readily converted into hæmoglobin. From milk and yolk he isolated such an organic combination. But he admits that inorganic iron preparations are capable of curing chlorosis, and explains this as follows. In chlorosis digestion is disturbed with formation of sulphuretted hydrogen and alkaline sulphides in the bowel. These combine with and separate out the organic iron of the food, and sulphide is formed, an inorganic compound which, according to Bunge, cannot be absorbed; hence the blood loses its necessary supply of iron, and chlorosis results. When inorganic iron is given, however, it combines with and neutralises the sulphuretted hydrogen, and thus protects the organic iron of the food, which, therefore, becomes absorbed, and goes to form hæmoglobin. In support of this view he adduces the received opinion that large doses of iron are necessary for the cure of chlorosis, and this he says is because large amounts are necessary to neutralise all the sulphuretted hydrogen in the bowel. Further, he states on the authority of Zander that hydrochloric acid cures anæmia more satisfactorily than iron does, because it is antiseptic, and prevents formation of sulphuretted hydrogen in the bowel." Now, as Stockman adds, "the presence of iron in our food, in the tissues and excretions of the body, its constant ingestion and excretion, and the small quantities with which we have to deal, apparently place a complete barrier in the way of rigidly proving by chemical methods that it is or is not absorbed."

Stockman met these difficulties by other expedients. First, in certain well-marked cases he removed the problem from the sphere of the bowel, and endeavoured to bring about the cure of chlorosis by injecting iron subcutaneously. Secondly, he administered sulphide of iron by the mouth, a preparation which cannot take up more sulphur, and, being non-astringent, cannot be credited with any tonic effect on the bowel such as might promote its absorptive activities. Thirdly, he administered bismuth, manganese, and other drugs which have a like power of neutralising sulphuretted hydrogen, and which should therefore have a like curative power in chlorosis. The results of these observations were as follows: both in his own cases, and in the cases of others who had given iron subcutaneously for other reasons, iron thus administered subcutaneously cured chlorosis, though the method is one which has its drawbacks; Dr. Warfvinge of Stockholm cured a series of cases by subcutaneous injection of iron, and found that thus used one-fifth of the ordinary doses of the metal sufficed: the cure of chlorosis seems then to be by absorption. Secondly, sulphide of iron proved a satisfactory means of cure. Stockman also emphasises, what many of us had noted, that reduced iron cures chlorosis in doses too small to have any substantial effect in neutralising sulphuretted hydrogen. I may add that patients have complained to me that reduced iron seems, indeed, to have the unpleasant property of disengaging sulphuretted hydrogen in the bowel, so that the drug is sometimes quietly shirked by the patient. Thirdly, Stockman found that bismuth, which would absorb even more sulphuretted hydrogen than iron, is nevertheless quite inefficacious in the treatment of chlorosis. Kletzinsky speaks, therefore, in paradox when he says that "from all the hundredweights of iron given to anæmics during centuries not a single blood-corpuscle has been formed." Stockman thinks that iron is absorbed as other salts are, the ferric salts being reduced to ferrous in the intestine; and that the building of it into organic combinations, which are of various degrees of intimacy, is done in the liver. Dr. Mackay stated at the Toronto Congress in 1897 that iron is absorbed by the epithelial cells of the villi, the iron of hæmoglobin being taken up as hæmatin. He adds that the metal is passed inwards by the leucocytes. Binz has stated, I think, that an early effect of iron in chlorosis is a multiplication of leucocytes. That they are increased in total number rather than drawn from their hiding-places would be difficult to prove; at any rate they may be more busily employed. That "inorganic iron" given as a remedy does no more than stimulate the atonic intestine to absorb the iron (Kobert) seems improbable. Bunge's ingenious suggestion seems, then, to be without foundation.

Toxic causes.—That chlorosis is due to the influence of some toxin in the system is a speculation which must have presented itself to many minds; and not a few pathologists have busied themselves with hypotheses of this kind, from the inevitable microbe to the mere absorption of fæcal juices from the constipated bowel, or the presence of uric acid in the blood (Haig). Bunge's hypothesis, indeed, rests upon some such postulates in respect of toxic agents, though in his view the toxins in the bowel act indirectly and within the canal. The toxic hypotheses of chlorosis depend for their proof on the discovery of such injurious agents in the blood or excretions. The simplest of them is that popularised by the late Sir Andrew Clark, who earnestly argued that the impoverishment of the blood in this malady is directly due to constipation of the bowels; this, he said, brings about an accumulation of the products of decomposition in the alimentary canal which, passing thence into the blood, poison it either in its prime or at its sources. Stockman, Simon, and other observers who have tabulated cases with this problem in view, point out that, in the first place, only about half of the cases of chlorosis present constipation; while, on the other hand, constipated people who do not suffer from chlorosis are common in both sexes. When Clark published his paper I paid close attention to this point, and accepted no mere routine reply to my inquiries into the state of the intestinal functions; and I likewise found reason to believe that, when the cases are excluded in which constipation is attributable to the iron administered, chlorotic women are not more constipated than other women. We shall see presently that this hypothesis of toxicity is not without considerable importance in the field of therapeutics; no one has even pretended to show that chlorosis is to be cured by purgatives alone, yet, on the other hand, I detect in almost all writers on chlorosis a temptation to rely on the toxicity of the blood in one direction or another. Even Lloyd Jones, believing as he does that chlorosis is but an abnormal intensity of a normal storage process, fortifies himself with an argument out of the same quiver; he has tested the ovarian system for such a poison, so far without success, and still has his eye on the uterus as an alternative source. Van Noorden (36) and Arcangeli are likewise disposed to assume some perversion, absence, or excess of an ovarian internal secretion as a factor in chlorosis. Chvostek reported that in twenty-one cases out of fifty-six he found the spleen enlarged; thirteen times it was palpable: thus he also is led to support the alleged kinship between chlorosis and splenic anæmia. Clement, if I understand him aright, looks for the toxin or infective agent outside the body; and, partly on analogy, considers that chlorosis should be classed with the infectious diseases. He tells the story of an epidemy which occurred in a small village, during which eight young girls were attacked with febrile symptoms and enlargement of the spleen; phlegmasia alba, dry pericarditis, and pleurisy were among the complications. Anæmic these patients were no doubt, but few readers will be convinced that the malady under which they suffered was chlorosis. Against these allegations of enlarged spleen I may say that Simon and Schrott, both of whom had their attention directed to this point, found this enlargement in one case each [*vide* "Spleen in Anæmia," vol. iv. p. 522; and art. "Splenic Anæmia," p. 539 of this volume].

Pick finds the source of the blood-poison in another place, namely, in a dilated stomach. His cure for chlorosis is lavage. Nothnagel takes substantially the same view of the matter as did Clark. Now, no doubt, certain poisons do reduce the blood; such poisons as lead, arsenic, syphilis, those of acute rheumatism, Bright's disease, and pernicious anæmia, and so forth; but it is a superficial way of looking at things to say that anæmia here and anæmia there must be due to like causes. If we are to listen to comparisons of this sort we must have the specific gravity of the blood-serum in all cases, and from it we must learn whether these anæmias are all of the same kind; that is, whether the blood plasma keeps up to the standard of health, or even rises above it, while that of the whole blood falls. Jones tells that such is the feature of chlorosis; and Hayem, Stockman, indeed all careful students, tell us that in experiments and observations on this subject regard must be had to the kinds of the changes in the blood. Chlorosis, as Immermann well says, "maintains its individuality in the teeth of all the attempts that have been made to merge it in the great ocean of anæmia."

Again, in anæmia there is, or often is, no evidence of poison in other parts or excretions of the body. For example, Simon (50) says that indican is not found in the urine of the chlorotic; and Rethers, by a series of important investigations, seems to have shaken the foundations of the toxic hypothesis, by showing that in 9 out of 18 cases of ordinary and severe chlorosis the ethereal sulphates were absent; and that in the remainder there was no uniform or considerable appearance of them. Von Noorden, who (p. 347) discusses this point clearly, quotes Hennige and Heinemann to the same effect; and Stockman adds the testimony of Mörner. The secret of the causation of chlorosis does not seem to lie, then, in a foul state of the intestine, or in the absorption of some poison; the evidence lies in the direction of diminished manufacture and metabolism rather than of accelerated destruction. The very poorness of the urine in many cases of definite chlorosis, its actual deficiency in colouring matter, indicates that, instead of an excessive breaking-down of blood corpuscles, such as results from the absorption of poisons of the kind under consideration, the life of the red cells is, on the other hand, prolonged.

There seems to be a certain, though probably not a very intimate, association between chlorosis and Graves' disease. Chvostek gives seven cases of chlorosis associated with Graves' disease. Lloyd Jones and others also note some fulness of the thyroid in many cases. I have observed the same coincidence; but without a calculation of the frequency of some fulness of the thyroid in healthy women it is not easy to express an opinion on the point. Professor Stockman supposes that chlorosis depends mainly upon two causes; namely, on insufficient food at the age of development, in which conclusion he is supported by Simon, and on the persistent effects of incidental hæmorrhages, menstrual and other, which may be positively excessive, or relatively excessive in the individual case. When, however, we regard the many contingencies to which the operation of these several causes are open, the partiality of their incidence, and the many cases in which these factors produce disorders other than chlorosis—such as mere emaciation and debility, with a fall in the proteid value of the blood—I repeat that, in my opinion, we have to look for a more uniform cause, one more independent of contingencies; such, perhaps, as that proposed, rightly or wrongly, by Dr. Lloyd Jones.

I fear Dr. Haig's uric acid hypothesis has little to support it; against it we find on all hands that the excretion of nitrogen in chlorosis is rather diminished than increased. Gräber, moreover, found the alkalinity of the blood up to the normal standard, and even above it; and Von Noorden tells us that Peiper, Kraus, Rumpf, and Dronin corroborate this statement.

Pathology.—In the discussion of the causation of chlorosis we have dealt incidentally with matters of pathology or pathogeny; the remaining part of the subject will not detain us long. On pricking the finger of a chlorotic patient, bloodless as she may appear, the blood springs forth freely, more freely than in anæmias of other kinds; the colour also is different; the red corpuscles being fewer, the blood transmits light more readily and the colour is brighter; it is bright red or even borders on orange. The specific gravity of the blood is easily tested by Roy's method, but that of the plasma less readily; for this a centrifugal machine is required. The specific gravity of the blood is reduced; that of the plasma is steady, or possibly even raised. Dr. Lloyd Jones tells us that the mean specific gravity of the blood rises in both sexes alike till puberty; at this period, however, that of the man still rises, while that of the woman falls. Taking the blood of childhood (two to three years) at 1050, that of a young man of seventeen may be 1058; of a young woman 1055·6. These observations have been made, of course, under dietetic and other controls. From the age of seventeen, then, Jones finds that the mean specific gravity in man still rises; in woman it remains low till twenty-five, after which age it rises to 1055 or 1056. Coincidently with these changes in the blood general metabolism is lessened, for the excretion of carbonic acid and of urea also falls (Landois and Stirling). In the Charts herewith, which I am enabled by the kindness of Dr. Lloyd Jones to publish, these changes are well exhibited. Whether the hæmoglobin be increased, unaffected, or decreased during or by menstruation seems as yet undetermined. I may repeat that Dr. Jones says, speaking generally, that the specific gravity of the blood stands at a lower mean in women who have many brothers and sisters; that indeed the specific gravity may be taken, approximately, as a gauge of fertility, the change in the blood in chlorosis being an extreme fluctuation of a physiological quality of the child-bearing period of life. Every girl, then, may be regarded as potentially chlorotic, and perhaps none passes through young womanhood without some phase of the disorder. The boundary between the physiological and the pathological states, if Lloyd Jones' conclusions are to be accepted, is an arbitrary one.

The specific gravity of the serum differs little, if at all, from that of health; if anything, it tends to rise. That of the blood falls as a whole by the diminution of the volume of the red corpuscles or of their hæmoglobin, usually of both; changes which are commonly recognised in chlorosis. In twenty-six cases tabulated by Jones (p. 22) the fall of the number of red corpuscles comes out strongly, so strongly as to teach us that this fall, taken together with a like fall in hæmoglobin value, is more characteristic of chlorosis than we are wont to suppose; at the same time the proteid value of the blood keeps steady. The alkalinity of the blood, especially of the plasma, as here said, is usually increased.

The reader thus perceives that the features recognised in chlorosis are the converse of those seen in some other forms of anæmia, such, for example, as ankylostoma and pernicious anæmia.

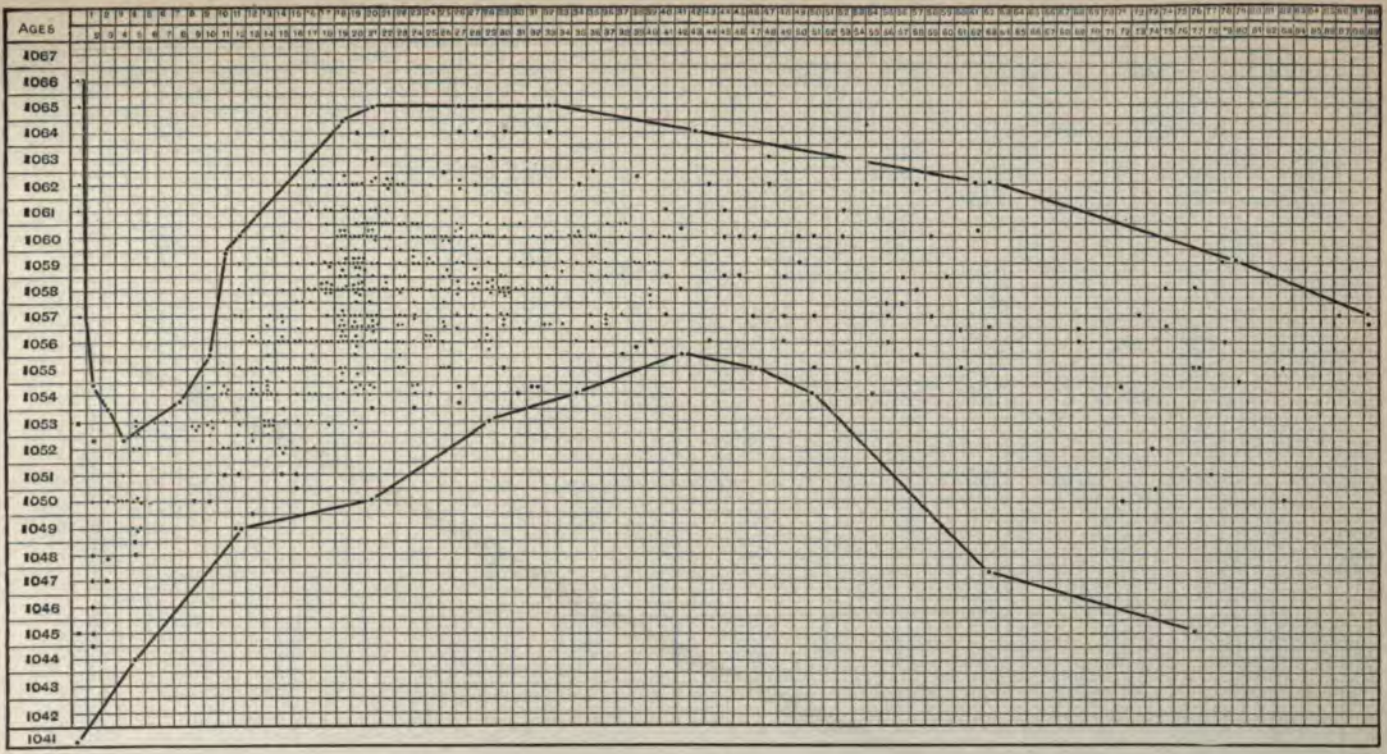

CHART 2.—Showing the results of observations on the specific gravity of the blood of healthy males of different ages, and the upper and lower limits of variations consistent with health. (Lloyd Jones.)

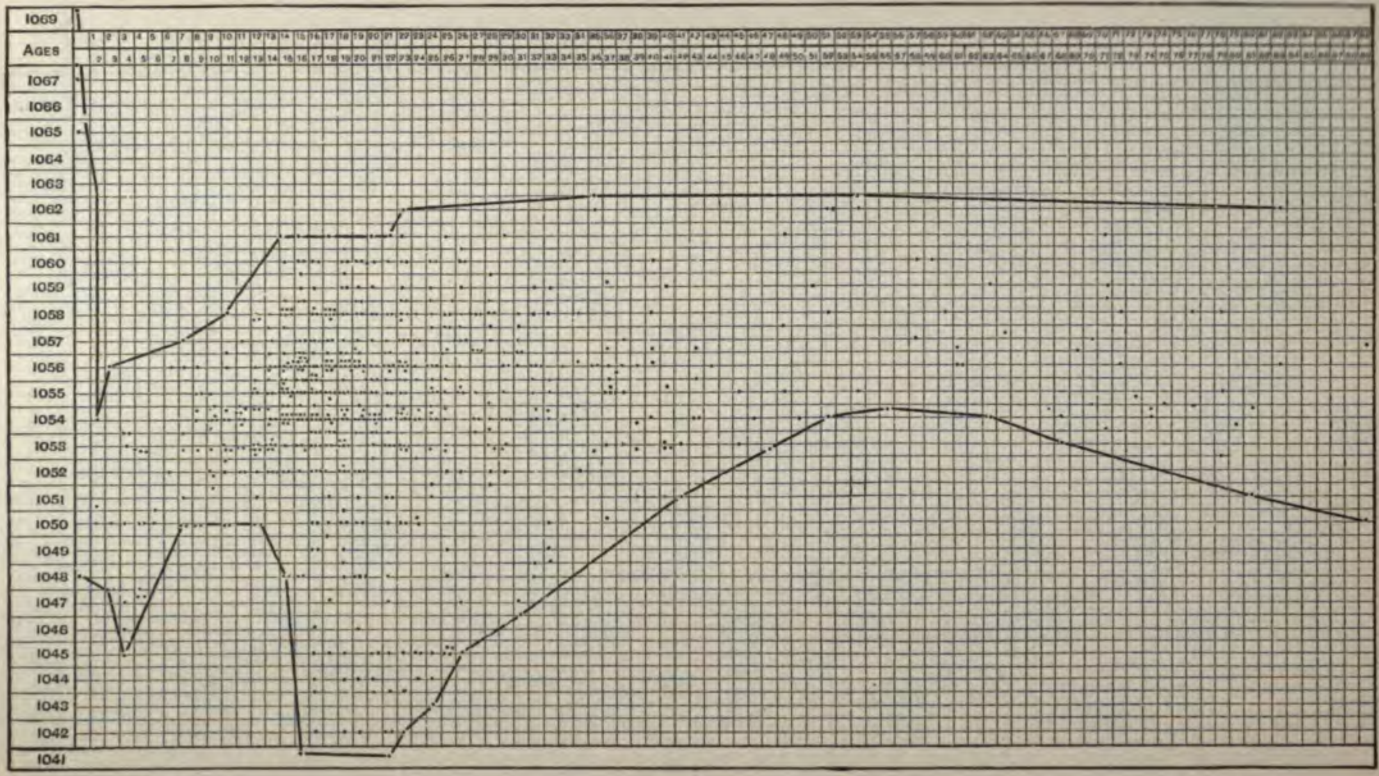

CHART 3.—Showing the results of observations on the specific gravity of the blood of healthy females of different ages, and the upper and lower limits of variation consistent with health. (Lloyd Jones.) The two charts represent observations on 1400 individuals.

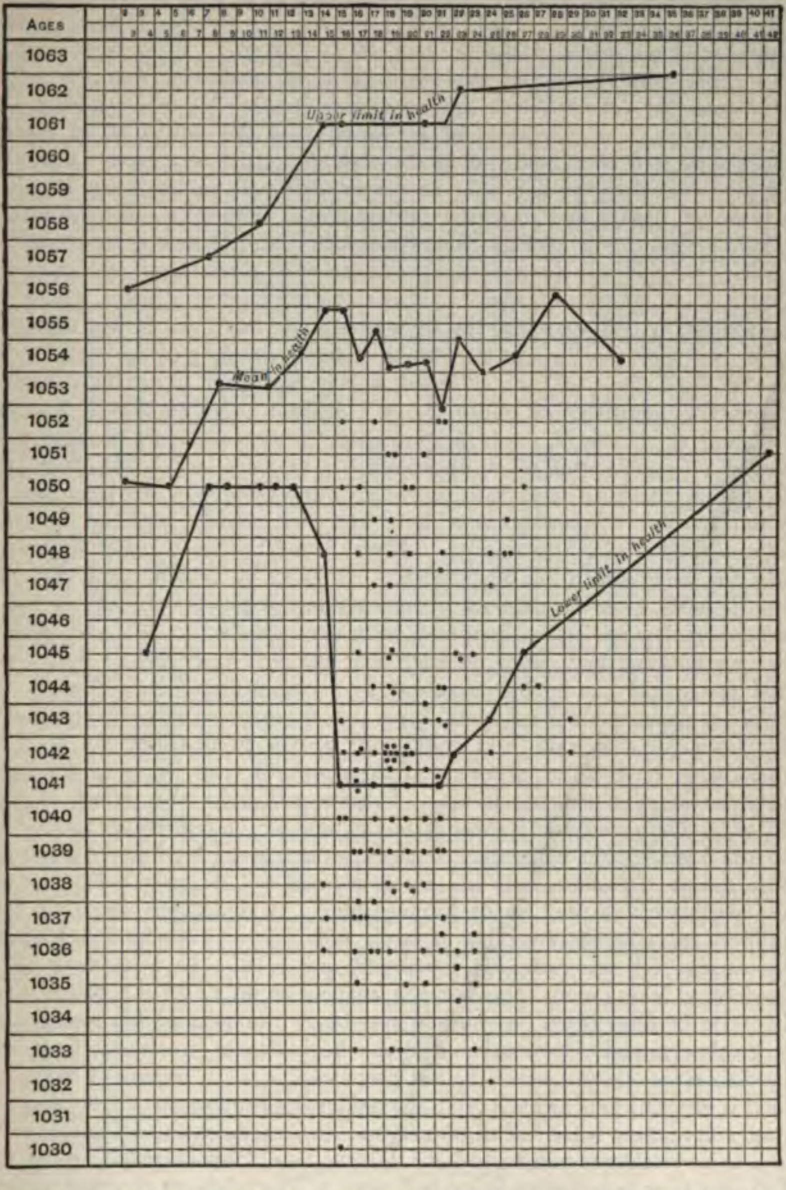

Chart 4.—Showing the variations in the specific gravity of the blood in healthy women, from 2 to 42 years of age, and the results of observations upon 120 young women with chlorosis. (Lloyd Jones.)

In Professor Stockman's no less careful inquiries the bearing of diet on chlorosis is estimated. That the iron needed for the blood is absorbed from the food seems probable, nay, it is proved. It is certain that the chick gets iron from the yolk, and the suckling from the milk. Stockman has estimated our sources of this metal. He takes the issue of iron daily as $\frac{1}{10}$ of a grain, and he found that the daily food of fifteen healthy persons contained iron at the rate of $\frac{1}{10}$ to $\frac{1}{6}$ of a grain; thus the supply is equal to the demand: moreover, detained in the liver, there is a store of disengaged iron, the precipitate of defunct corpuscles, which acts as a reserve; in healthy livers Stockman found from $2\frac{7}{10}$ to $4\frac{3}{5}$ grains of it. Now if we compare this estimate with the state of chlorosis we find a derangement of these relations. Although in women, owing to menstruation and so forth, the reserve iron in the liver is less than in men, yet their food is apt to contain a smaller supply of the metal. In the habitual diets of four chlorotic women Stockman found iron in the respective quantities of $\frac{1}{30}$ to $\frac{1}{20}$ of a grain a day; so that if iron be escaping at a rate of $\frac{1}{10}$ of a grain daily, the red corpuscles may well starve. Coppola and other observers fed animals (cocks and dogs) on food from which all iron had previously been removed; the hæmoglobin value of the corpuscles soon fell, but was readily replaced on the administration of inorganic iron. It was found that on a non-ferruginous diet the hæmoglobin fell 35 per cent. It is to be remembered that the iron is held in various degrees of intimacy in the articles of diet; in some organic molecules, as, for example, in the protoplasm of cells and nuclei, the combination may be so intimate that ordinary tests fail to detect it; and the metal has to be recovered by incineration (Zaleski, Vay). In ordinary anæmia of an accidental kind—as, for instance, after an occasional hæmorrhage—the food iron commonly proves to be sufficient for the restoration of health; although, if 20 grains of iron be lost, the food may be long in making it up. I have said that the defect of the blood in chlorosis usually extends to the number of corpuscles as well as to the hæmoglobin contents. Both Stockman and Lloyd Jones insist on this defect; Stockman, in his cases, reports a "striking deficiency of red corpuscles as well as of hæmoglobin." A deficiency down to two millions may be observed, but it rarely falls lower. Stockman's highest figure was 66 per cent, his lowest 20 per cent. In four days after beginning ferruginous remedies the corpuscles go up with a bound, and in 10 or 14 days reach the normal standard; the hæmoglobin rises much more slowly, and two months after the beginning of treatment may still be defective. Deformity of the red corpuscles is not a feature of chlorosis as it is of pernicious anæmia; but a considerable proportion of them may be under-sized, even when full allowance is made for the presence of microcytes, and their vitality is said to be low; that is, their histo-chemical properties fall, and they fade before doing full work. The relations of water and of salts to the serum of the blood are less easy to discuss: the steady specific gravity of the proteids in the plasma would indicate that there is no tendency to hydræmia. It seems probable that the amount of water stands in some definite relation to the proteid constituents, as its presence is not a mere dilution, but a combination with these substances: in like manner the salts are related to the state of the proteids and to the cell activities. For the present, however, it does not seem that these are points of primary importance in chlorosis.

Coagulation is slower in chlorotic blood, outside the body, notwithstanding the tendency to thrombosis within it; the clots are scanty, and the fibrinogen is less, facts which are not easy to reconcile with such an accident. A slow venous blood-stream may account for the tendency to thrombosis.

We can only guess at the *mass of the blood* in any case; in many anæmias we have seen that the mass of the blood seems to be diminished, the blood issues slowly from a puncture; in chlorosis the fulness of the vessels seems to point to a persistence of the normal blood mass, and such is the assumption of most writers. Dr. Lloyd Jones thinks that the dyspepsia of chlorosis is due to an accumulation of blood in the great veins of the abdomen, the dilatation of which he attributes to some influence on the splanchnic nerves. If there be an increase in the mass of the blood, there may be an actual "plethora serosa," with or without hyperalbuminosis. If the aorta be small, there may be a relative plethora. In some cases of prolonged chlorosis there may possibly be some mesoblastic hypoplasia of a transient kind, and the capacity of the arterial tree may grow with its nutrition and its contents. In ordinary cases, however, the plasmatic elements of the blood seem to be sufficient for vegetative growth; chlorotic girls do not lack size, nor do they fall away from the main lines of development.

On the other hand, the heart and vessels are subject to deterioration of a somewhat serious kind. The arteries, and especially the aorta, near the origin of its ascending portion, may present on the inner coat dull yellow spots and striæ; indeed, superficial erosions of some extent may be detected. The striæ may also be seen in the descending portion on its posterior inner aspect, between the intercostal and lumbar arteries (Virchow). These patches and streaks, when examined microscopically, are found to consist of minute dots, each, according to Virchow, being a fatty connective tissue corpuscle. The heart is commonly of normal size, but it may be moderately dilated. Valvular disease is rare, as is ordinary atheroma; but fatty degeneration of the heart is a feature of chlorosis, as of all anæmias. The change is primary, and is best seen in the papillary muscles, especially of the left ventricle, as spots and striæ; healthy fibrils being mixed with fatty. Virchow also describes fatty degeneration in the capillaries.

The renal, hepatic, and gastric cells are fatty also, and all the organs are pale. The atonic stomach may be dilated. The spleen, marrow, and lymphatic glands are not understood to be abnormal.

Symptoms.—The chlorotic girl is known in every consulting-room, public or private. The disease is no respecter of rank or of fortune. Whether her aspect at first sight be indicative of the disease or not, her characteristic complaint is dyspnœa. Dyspnœa, due probably to incessant stimulation of the bulb by suboxidised blood, is more persistent and incapacitating in chlorosis than in any other disorder, except, of course, in advanced organic disease of the heart. Many of these patients bear in their features the classical sign of their malady, but not so all of them; many of them carry some colour (chlorosis florida); but in my experience all suffer from dyspnœa, and, however insidiously it creep on—for the disease may attack acutely or insidiously—the patient is never unaware of it. If she be asked whether she can trip upstairs as she was wont to do a few months previously, her answer will bring the physician near to his diagnosis. I have said that many chlorotic girls carry some colour, indeed a high colour. In past years this was a little puzzling to me, as no doubt to others also; but we have now learned to look below the surface, and I see that Stockman, Lloyd Jones, and many others deal with this aberrancy, and point out why even a high colour may not be incompatible with chlorosis. It is said that the conspicuous chlorotic is a blonde; but surely we see many blondes who in spite of an assured chlorosis have some carmine in their cheeks, and many brunettes who are pale enough and green enough to reveal their disorder. Much depends, I suspect, upon the skin of the individual; a fair and clear skin takes the alabaster or old wax colour, a brown and a muddy or thick skin does not. We have blondes with bad complexions and brunettes with transparent complexions. Transparent skins are often seen in the dark women of the so-called Iberians among ourselves, and chlorosis is manifest enough in them; on the other hand, I know many blondes who have not presented the standard tint of chlorosis, although suffering from it in no slight measure: in such persons a thick complexion conceals or modifies the characteristic tints; grayness or sallowness takes the place of marble or alabaster, and chloasmic tints may be seen here and there on the temples and about the knuckles and other joints. Such women do not flush readily or deeply, and their limbs, often rough and hairy, do not offer a good surface for the display of the changes of the blood. The upper part of the chest, bared for the stethoscope, may, however, manifest the peculiar hue; a pallor may be detected also in the lachrymal caruncle, on the under side of the conjunctiva and the mucous membrane of the mouth; the sclerotics may be blue, the pupils dilated, and the ear—that useful signal of variations in the colour of the blood—may be white. The nails, also, and the blue veins on the skin may have their story to tell.[1] The pupils are often dilated, brightening the eye, though the face is often described as inanimate and puffy; perhaps this word "puffy" is not a very accurate one. If chlorosis be a disease in which the proteids of the blood are not wanting, and if the mass of the blood be not diminished, the face may retain its ordinary contours and yet seem, as it were, puffed by contrast with other signs suggestive of serious ill health. In this kind of chlorosis epistaxis may occur, even profusely. It is preceded by a sense of fulness and discomfort, and is followed by a sense of relief. In the anæmias of malaria, plumbism, or cancer, the vessels are more empty and the face more shrunken. Chlorotic girls still blush readily enough, and even in the height of an attack of their malady some of them never lose a vivid carmine on the malar eminences. There is another peculiar sign to be seen in the face: Dr. Lloyd Jones says that if a healthy person be asked suddenly to look up at the ceiling, without moving the head, the eyebrows are raised and the forehead is thrown into horizontal folds by a contraction of the anterior portion of the occipito-frontal muscle. Now, in many chlorotic women this associated movement of the occipito-frontal muscle is wanting; as Joffroy pointed out also in Graves' disease: yet they can contract this muscle if they try to do so. Lloyd Jones attributes this lagging to a lessened irritability of the skeletal muscles, due to lack of hæmoglobin; and thus it is that the face appears inanimate or even apathetic, the languid, listless look of chlorotic patients being partly due to want of facial expression, partly to slackness of the limbs (Hayem). Sydenham says that "crurum tensiva lassitudo" is a complaint of these patients. Yawning betrays a like muscular affection, and to the patient is often a troublesome symptom.

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[1] For the following ingenious method of measuring the oxidising activity of the blood in chlorosis I can say nothing of my own experience: I therefore put it into a note. The passage is translated and a little abbreviated from Henocque, "L'hematoscope," *Gaz. hebd.* Oct. 23, 1886, and April 1, 1887. It is quoted by Gilbert (10).

The reduction of oxyhæmoglobin into hæmoglobin in the tissues can be determined by spectroscopic examination of the blood through the thumb-nail. Thus the first band characteristic of oxyhæmoglobin may be seen, sometimes the second also. If a ligature be tied round the phalanx the bands disappear, the yellow on the level of the line D reappears, which was concealed, and then the bands vanish. The ligature isolates in the thumb a certain quantity of oxygenated blood, which for a certain time exhibits the bands of oxyhæmoglobin; the latter gives off its oxygen to the tissues, is reduced, and the absorption band is no longer intense enough to traverse the nail. In the normal state this process occupies 70 seconds, and the quantity of oxyhæmoglobin thus reduced is 0·20 per second. This is taken as the unit of reduction. In chlorosis the oxidising activity falls to 0·65-0·19 of this unit; the mean fall being 0·44.

Digestive system.—The tongue is pale, moist, indented, and often clean. At other times it presents on its coated surface some evidence of disorder of the stomach or associated organs. The breath likewise in some patients is heavy in odour. Such patients are often constipated, though great constipation is consistent with a clean tongue. That constipation, however frequently seen, is not by any means a constant symptom I have already pointed out. It seems to be noted in about one-half of tabulated cases.

The stomach is often the seat of some morbid changes; it may be permanently dilated, though more often temporary distension and atony may simulate such dilatation. I am not prepared to say that dilatation in the formal sense of the name is so common in chlorosis as to make a part of our ordinary conception of the malady (vol. iii. p. 494); nor do I regard it as a very common complication: were it so, the cure of chlorosis would be a matter of more serious difficulty than it is. Dyspepsia of the subacute or chronic catarrhal kind, or that of flatulence and atony, sometimes stops the way, but it rarely defies the usual means of treatment. The appetite may give us more trouble. I have said that it is often marked by caprices and perversions which put serious obstacles in the way of nutrition. At the same time these symptoms are seen in other states, such as neurasthenia—the dislike of meat especially—and are, perhaps, characteristic rather of the kind of patient than of the kind of disease. In neurasthenia this anorexia or parorexia leads to emaciation; in chlorosis this is not generally the case. In anorexia nervosa (Gull) the wasting is also remarkable; but the chlorotic woman eats more: her blood is also richer in proteids, and being short of oxygen permits the deposit of fat. The relations of chlorosis to ulcer of the stomach are dealt with in the article on the latter subject (vol. iii. p. 519).

Diarrhœa is a rare and incidental occurrence. Hysterical chlorosis is more compacted of fantasy, bizarrerie, and caprice than the common and uncomplicated form of the disease; or tears and melancholy may alternate with fretfulness and self-importance. "Hysterical or barking cough" is a very trying feature of these cases; indeed, I have found it a not infrequent feature of chlorosis not otherwise marked by hysteria or neurosis. Such coughs may be interpreted by an examination of the blood, and cured by iron; for indeed they are rather chlorotic than hysterical in nature.

Circulatory system.—Sydenham describes "pulsus febrilis" as a symptom of chlorosis; and later authors, relying on the thermometer, describe a "febrile chlorosis." Were not such observers as Prof. Osler in their ranks I should be content to say that "febrile chlorosis" is an aberrant form of the malady, and the fever may prove to be significant of some complication. The use of such a name by less experienced physicians might, for instance, lead to confusion between chlorosis and pernicious or syphilitic anæmia, or some other anæmia due to a toxic agent. I should be content to say that the temperature in chlorosis is not subnormal, as in anorexia nervosa, for instance; and that it may be apt to rise in trifling measure under the influence of occasional causes.[1] The pulse, however, as Sydenham said, is generally quickened more or less, and is very impressionable by change of posture and the like. In opposition to some authors, I am scarcely disposed to admit that in chlorosis the arterial blood-pressure generally ranges above the normal standard; though no doubt it is characteristic of chlorotic anæmia that such a rise may be observed occasionally; and as a rule the mass of the blood is not diminished, the artery is well filled. Immermann seems to have given vogue to the opinion that in chlorosis the heart increases and arterial blood-pressure rises. Bihler, on the other hand, who has gone over this ground carefully, concludes that so far from the blood-pressure being raised it is usually under the normal mean. Estimations of blood-pressure made with the ordinary sphygmographs are of little or no value. As, generally speaking, the mass of the blood is not diminished, the output of the left ventricle is at least normal in amount, and the arteries are well filled; but this does not necessarily or even probably mean increased blood-pressure. Extension of cardiac dulness is generally towards the right, not towards the left side. As vascular tone increases, the heart returns to its normal limits. Chlorotic women are liable to syncopic attacks which seem to indicate that the blood-pressure, if fairly sustained as a mean, is nevertheless subject to great or extreme variations. The arteries often throb in chlorosis; although often these vessels are really full, they are much slackened in tone: we may commonly see pulsation in the epigastrium and in the episternal notch, and both first and second sounds are very loud in the carotids. The second sound is often louder at the apex than at the aortic cartilage.

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[1] Dr. Stockman tells me later that he finds that all bloodless people are liable to slight febrile attacks. The cause of this instability is discussed by Gräber and others.

The heart is irritable; it often palpitates, it may be to the great distress of the patient. The palpitation makes itself felt rather on exertion—on the least exertion; a perturbation due to the call of the anæmic tissues, probably the muscles, for more blood, that is for more oxygen; their supply of proteids is probably sufficient. The heart itself, on examination, is found to vary a good deal. Although its beat is often throbbing or laboured, yet not less often it is feeble and ill-defined. That the heart is dilated is the assertion of many observers; however, the facility with which modern physicians delineate and record the varying dimensions of this organ excites my admiration of a skill I cannot hope to attain; the conditions of physical diagnosis seem to me indeed to be too inconstant for such appraisements. For instance, in this and other ailments of young women I have observed (cf. vol. iii. p. 505), and the observation is by no means confined to myself (34), that the mean volume of the lungs is often reduced. The respiration in chlorosis is obviously shallow; and, although to tight lacing is attributed every mischief which may befall a woman, yet it is indeed probable that the fashion of feminine garments prevents the full excursion of the diaphragm; thus in chlorosis the lungs may shrink, and the heart more or less denuded may offer a larger front to the auscultator. In cases of alleged cure of dilatation of the heart we may have a contrary phase; the lungs may expand under this measure or that, and the heart be enveloped over a larger part of its surface than before: in view of such changes as these it seems very difficult confidently to infer, in the one case or the other, that the organ is much altered in size and shape. That in anæmia, generally speaking, dilatation of the heart as of the stomach is prone to occur from loss of tone I have already said, but that in chlorosis the heart undergoes an enlargement both in substance and in capacity is not proven. We know, indeed, from the appearance of apex murmurs and the tendency to fatty degeneration, that the organ may yield a good deal; yet a case of chlorosis must be of extraordinary severity to bring the patient to the post-mortem table. Over-exertion under such cardiac conditions may cause "irritable heart" or "weak heart"—symptoms, by the way, which the ailing patient may never get rid of.

Venous murmurs.—The murmurs heard in the heart and veins in chlorosis have been studied with an interest enhanced by the obscurity of their causation. The phenomena are very common, they are demonstrated to every student in the out-patient room, and the problem of their generation is a fascinating puzzle for every ingenious clinician. And whoso cannot himself explain can select his explanation from the teacher whose doctrine he prefers.

The venous hums, which, although they may occur in any anæmia, are very characteristic of chlorosis, may be considered first. These murmurs—known as *bruit de diable* by the French, as *Nonnengeräusch* or *Venensausen* by the Germans—the two former names being taken from the humming-top—are most often heard in the jugular veins, usually more loudly on the right side. The sound in the jugular vein is a persistent hum, likened by Sansom to the shell sound which Landor has made his own; Sir Thomas Watson likens it to the hum of a gnat or to that of the wind sighing through a crevice (47). When this hum is loud it can be felt; if the left hand be laid on the neck, grasping it lightly so as to let the thumb rest upon the right jugular, a vibration in the walls of the vein is perceptible to the touch; and by such pressure on the vein as shall stop the venous current the hum is made to cease. It is heard best in the standing position, being favoured by gravitation; and during inspiration. It is clear, then, that the hum is generated in the vein. If the patient be directed to take a deep breath, or to rise from a recumbent to an upright position, the venous current is accelerated and the hum is intensified. The sound is usually louder in the right jugular, because this vessel, by way of the innominate vein, enters the vena cava almost in a right line; whereas the left cervical veins collect and fall into this channel at a considerable angle. Under these and other circumstances the pitch and intensity of the murmur may vary. For the same reason it is sometimes louder during the cardiac diastole; and if, instead of suppressing the sound by stopping the vein, the stethoscope be very lightly pressed on the vessel the murmur may be increased. To turn the head to the opposite side may have a like effect; but the sound is a capricious one, and that disposition which on one day or in one person seems to intensify it, on another day or in another person may extinguish it; often indeed it varies extremely while under continuous observation. It is not difficult to suggest an explanation of the hum; that which is generally given, and which on the face of it seems most probable, is that the vibration of the walls of the vein is due to a change in the calibre of the tube at the root of the neck. The lower portion of the vein is of constant or almost constant calibre; this constancy being secured by the adhesion of the coats to the cervical fascia. Now if by any means, such as a smaller stream of blood, the vessel be narrowed above, there is a run of the blood from a narrower to a wider channel, this change in the continents sets up fluid veins in the contained blood, and the walls of the vessel are thrown into vibration thereby. Still, although this explanation is rational in itself, yet we may ask why it occurs in some anæmias and not in others? Again, why is it—as I think it is—incomparably more frequent in chlorosis than in other anæmias? In my experience it is not usual to get the venous hum in plumbism, in malaria, in cancer and so forth; it may be there, but it is not to be foretold, while in chlorosis to foretell it is a fairly safe prophecy. Yet if it be true that in chlorosis the vessels are not empty as they are in some other anæmias, surely it is in chlorosis that the hums should be less commonly heard. Perhaps the tone of the vessels enters into the causation. Moreover, there is an old hypothesis that the hum is due to the "thinness of the blood," the corpuscular contents of which as we know are notably reduced: this hypothesis has never received much countenance from competent judges; but Potain has brought it forward again on the basis of experiment. Potain so arranged a tube in connection with a reservoir that at one time serum should run down the tube, at another defibrinated blood containing the normal number of red corpuscles; on the use of the stethoscope the murmur was heard to fall in intensity when corpuscular blood replaced the serum. Whether this observation has been verified by other observers I do not know; if so, it has an important bearing on the generation of the *bruit de diable.* The hum, as I have hinted, is to be heard less certainly and loudly in other veins, in other kinds of anæmia, and even in some healthy persons. Many years ago in a foreign hospital I was told to hearken for a murmur on placing the stethoscope on the eyeball of a chlorotic patient; by this manœuvre, which I have often repeated since, the hum, fainter than in the jugular, can be heard; but before we can say that it is generated in the cerebral sinuses we must be sure that it is not transmitted from the jugular through the bones of the face. Dr. Stockman tells me it may be heard sometimes over the torcular Herophili.

Cardio-arterial murmurs.—That a systolic murmur is not infrequently heard over the subclavian artery, especially on the left side and towards the outer third of the clavicle, is an old observation which has interested both elder physicians who have found food for speculation as to the modes of its causation and younger practitioners who have been alarmed by what they regarded as a sign of aneurysm. This murmur was carefully studied by the late Sir Benjamin Richardson, but I am not able at this moment to put my hand on the reference. Richardson named the murmur the "carpenter's murmur," as it is not uncommon in these and other labourers. To pursue this side of the subject would lead us into digression; but in chlorosis and other anæmias such systolic murmurs are to be heard in more than one artery. It is a matter of doubt, indeed, whether the systolic murmurs of obscure causation heard about the base of the heart in chlorosis are formed in the heart proper, or in greater or lesser part in the large vessels about the same region. Dr. Sansom offers the explanation that under nervous (vaso-motor) disturbance the arteries may be unequally affected in their calibre, some lengths being contracted, others dilated or of normal size; so that the blood passes from narrower to wider channels. If this be so, we are in possession of a *vera causa,* whether it be the actual cause or not. Richardson attributed the murmur in the subclavian, increased by manual labour, to the constricting pressure of voluminous muscles on the vessel; but as it may be heard in anæmic persons whose muscles are far from voluminous, we may find in Sansom's hypothesis an essentially similar explanation. For in anæmia, not in chlorosis only, the murmur is to be heard in vessels, such as the carotids, not mechanically constricted from without as in muscular men the subclavian may be. The sound may be generated also in Graves' disease. Sansom quotes from Roger a case in which this murmur was musical, audible at a distance from the body, and in every accessible artery of the body. No pressure of the stethoscope was needed to bring it out, and the persistent noise was a torment to the patient. It seems probable, then, that these sounds, like the venous hums, are due to vibrations of the walls of locally constricted vessels; and as they are but clinical curiosities we may not spend any more time upon them.

Certain murmurs heard in the region of the heart are of more importance. The humming-top sounds are little more than curiosities, as they cannot be relied upon even for diagnosis; but the heart murmurs, if such they be, may have a more serious signification. Physicians do not hesitate to say that some at least of the murmurs heard about the heart in chlorosis are mitral in origin, and significant of the deterioration of the cardiac muscles which we have already considered (p. 501). It seems clear, however, that more than one kind of murmur is to be heard in or about the chlorotic heart; and, if possible, these are to be distinguished, for some of them may be of a graver character than others. I am enabled by the kindness of Dr. Sansom to reproduce the useful diagrams published in his valuable work on the *Diagnosis of Diseases of the Heart*, wherein these problems are carefully discussed. The diversity of explanations of the cardiac murmurs of chlorosis, suggested by eminent observers, makes it difficult to treat usefully of the matter except from the mere phenomenal point of view; the moment we pass from phenomena to explanation we find ourselves not only in the midst of conflicting hypotheses, but also without any clue to a decision.

A precise appreciation of the phenomena is, then, our first duty. The murmurs to be heard in or about the heart are as follows:—(i.) First in frequency are the murmurs to be heard in the region of the pulmonary artery and conus (Sansom's diagram, Fig. 27). In my student days all murmurs of chlorosis heard about the upper chest were indiscriminately referred to the aorta; to Walsh, I think, we owed the closer description of these sounds with which we afterwards became familiar. All recent observers are agreed that the murmur now under consideration occupies the area delineated by Sansom; and Sansom says that it is "greatly influenced" by the posture of the body, being louder as the patient returns to the recumbent attitude. This reinforcement is largely due, no doubt, to the retardation of the pulse-rate. In this quality it is to be distinguished from the organic systolic murmurs most of which are less influenced by this change. Dr. Sansom quotes Handford (14) to the effect that this murmur again increases as the patient turns over to the right, and wanes as she turns prone on the face. It varies with respiration, but in no constant way. It is to be remarked that in these cases pulsation is often to be felt about the parts occupied by basic murmurs, namely, in the second and third intercostal spaces, or even lower, and in the episternal notch. This we have all often observed and demonstrated at the bedside.

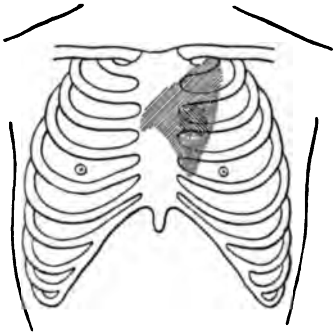

FIG. 27.—Area of pulmonary artery and conus, 59 per cent of cases. (After Sansom.)

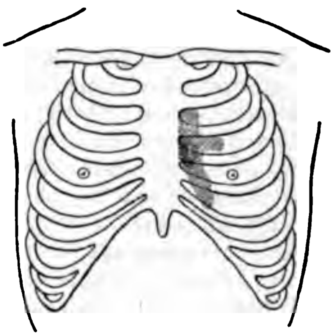

FIG. 28.—Area of right ventricle and conus, 11 per cent of cases. (After Sansom.)

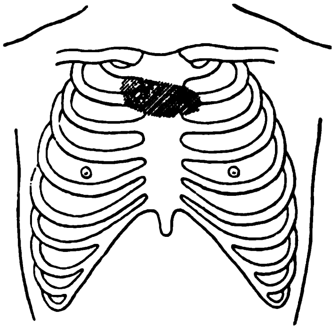

FIG. 29.—Area of aorta, 11 per cent of cases. (After Sansom.)

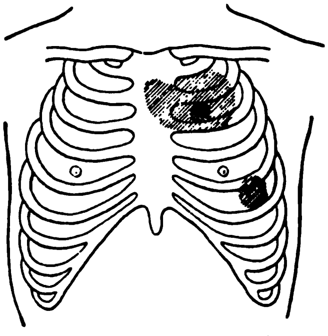

FIG. 30.—Systolic murmurs in pulmonary artery and at apex coexisting, 9 per cent of cases. (After Sansom.)

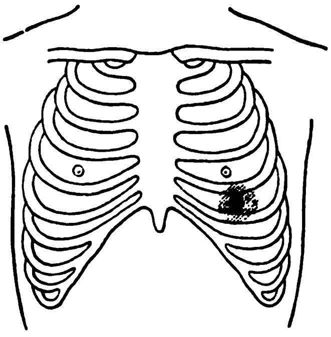

FIG. 31.—Systolic apex murmur only, 7 per cent of cases. (After Sansom.)

Now in respect of this pulsation we shall remember that in Graves' disease, where these pulsations are very evident, we also hear these basic or "pulmonary" murmurs, although the blood may present no change in the red corpuscles, either in number or colour. Sansom gives a very definite account of these murmurs; he says that in twenty-nine of his own cases murmurs over some part of the cardiac region were heard in sixteen; and in eleven they were in the pulmonary area. There is a large amount of evidence that similar murmurs may be produced by displacements of an otherwise normal heart: one such case I remember well in which, after death, the absence of all cardiac mischief was verified.

Arguments of weight seem to prove that these murmurs about the pulmonary area are not due to mitral regurgitation (Balfour), nor to pressure of a dilated auricle on the pulmonary artery (Russell, Handford). Dr. Sansom conjectures that the murmur is due to want of apposition of the mitral flaps on account of an enfeeblement of the muscular apparatus of the left ventricle. This is a modification of Balfour's surmise, and open to similar difficulties. I lean to the belief that the solution will be found in some altered relation between the blood and the walls of the vessels, especially the pulmonary artery and conus arteriosus; so that an excessive vibration of the walls takes place; if this be so, the cardiac murmurs, or some of them, will fall into line with the arterial vibrations of the same disease (p. 503) and with the venous hums. The pulsation of the vessels felt under other like conditions seems to lead us in the same direction. In Graves' disease this vibratile state of the great vessels is apparent enough.

(ii.) The murmur the site of which is indicated in Sansom's Fig. 28 need not detain us long. No doubt it is substantially the same murmur as the last mentioned, diverted a little in its area by incidental circumstances which may be guessed at rather than known. Sansom found it in eleven per cent of his cases.

(iii.) The next murmurs to be dealt with are those heard about the aortic region; that is, at the base (manubrium sterni) and at the second intercostal cartilage (Fig. 29). These murmurs are soft in quality and diffuse, not leading in any certain direction. Seeing that we were formerly taught that a murmur at the base is a common feature of chlorosis, it is curious to hear from Dr. Sansom that in his series this was the rarest of the chlorotic murmurs. From my own impressions I am prepared to coincide in his opinion. I have also noted that a murmur may be heard in this area as distinguished from that of the pulmonary region; the two, however, may coexist, and indeed may be mapped out separately. Within a few hours of writing these lines I have seen a case of chlorosis, mild in degree, in which, with the venous hum and some arterial vibration, a systolic murmur was heard at the second right cartilage and in the episternal notch. There was no trace of murmur in the pulmonary area. The history of the case, as a rule, will prevent any confusion between this murmur and a murmur of organic disease, whether due to rheumatism or to degenerative changes; and a persistently anacrotic pulse is decisively in favour of organic disease.

(iv.) Finally, there is the apex murmur in the region indicated in Figs. 30 and 31. This murmur was found by Sansom in sixteen per cent of his cases, and is a more serious matter, for it indicates mitral regurgitation; though in the cases we are considering the disorder is usually of a curable kind. There is no experience of the kind to which we may look back with more satisfaction than to systolic apex murmurs, which in their characters corresponded in all respects with those of permanent organic disease, but which disappeared entirely nevertheless. Loud or harsh murmurs in this place are not so common, if I may speak for my own experience, as the softer murmurs; still, soft or harsh, they arise under like conditions of atony, and to our repeated surprise—for repetition does not do away with the wonder of it—clear away altogether on appropriate treatment. These murmurs, indicative of mitral regurgitation as they probably are, I have frequently heard in middle-aged men who have indulged too freely in the pleasures of the table; men who show perhaps a little sugar in the urine for a time, or other such sign of slackened health. The like murmur arises in Graves' disease, in pernicious anæmia, after hæmorrhage in childbed, and so forth. A certain lecture, published by Dr. Donald MacAlister in 1882, seems to me to throw light upon this subject. "If an animal be bled till it is feeble," he says, "a murmur indicative of regurgitation from the ventricle is heard with the heart sounds. You may inject proper salt solution to make up the normal quantity of circulating fluid, but still the regurgitation occurs. As the animal makes blood again, so that its muscles are properly nourished, the murmur disappears." On the clinical side such instances are to be culled on all sides from medical records; some of those recorded by Dr. Sansom, as progressing to dropsical and other systemic changes and yet to recovery, being among the most remarkable. Dr. MacAlister gave in his lecture what seems to be the explanation of this phenomenon; and about the same time he showed to me a small cast of the interior of a heart in systole which carried conviction on the face of it. Relying in part on his own observations, in part on those of Ludwig and others, Dr. MacAlister demonstrated the large part taken by the auriculo-ventricular muscular structures in closing this orifice during the systole. On inspecting the model, one began to wonder whether valves were not luxuries rather than necessaries; for the sphincter fibres, contracting during the systole of the ventricle, seemed to reduce the orifice almost to an imperceptible chink. This of course is not quite the case, for Hesse has shown that the amount of reduction thus attained is only about one-half of the expansion area. We have, then, to call in the known factor of muscular atony in anæmia to explain that mitral regurgitation is very likely to take place; the relaxed muscle fails to do its share of the work, and the valves cannot quite make up for the defect. Moreover, we know that the papillary muscles are among the first to suffer in impoverishment of the blood, and that in those cases of anæmia which, by their severity, bring the patient to the post-mortem table, these parts, vital as they are, are found in states of more or less fatty degeneration. It is reasonable to assume, therefore, that these muscles are slackened. The difficulty is to understand why dilated hearts occurring in elderly folks and under other cognate conditions are so often unattended by a systolic murmur.

In the section on Mitral Stenosis in the following volume Dr. Sansom will discuss the coexistence of chlorosis and the former disease. In this connection I have only to suggest that as both of these diseases are found especially in women a large proportion of coincidence must be allowed for.

Potain endeavours with much ingenuity to prove that the murmurs of anæmia, or the chief of them, are of pulmonary origin. It is impossible to do justice to Potain's views in this place, and the advanced student is referred to his memoir (39). Sewall states that all "non-organic" murmurs at the base of the heart can be stopped by pressure with the stethoscope.

Œdema of the ankles and feet is often very considerable in chlorosis, and occurs earlier than in other anæmias; as, for instance, of phthisis or cancer, when it is a sign of dissolution. In chlorosis it seems to bear little relation to the apex murmur, which may be present or absent. This subject is more fully and broadly discussed in a later article, on Dropsy. Although, as I have said, recovery may be anticipated with some confidence from these conditions, attended by murmurs, and even by further evidence of cardiac failure which up to a certain point we may regard as transient, it is an interesting point to decide when the murmurs and other symptoms indicate more than a dynamic change—when the heart disorder has entered upon an altered static phase. The hope of complete recovery need not be bounded by the appearance of dropsy; in many cases I repeat that all such symptoms have passed away entirely. I remember having a serious difference of opinion with a medical man whom, unfortunately, I had no opportunity of meeting personally, in respect of a case of chlorosis in a young lady in whom a mitral murmur was audible at the apex, and in the axillary and subscapular regions. Her own medical man had assured the parents that the chlorosis was but a subordinate matter, and a permanent heart disease the principal evil. For this malady she was put under conditions which were not in all respects good for the anæmia, including the mental distress thus entailed on the patient and her friends. I did my best to root out this disheartening prepossession, but with little immediate success. However, I accidentally heard, a year later, that the subject of incurable heart disease was playing lawn tennis vigorously at all the parties in her neighbourhood. Yet I would not lead the unwary reader to mistake, let us say, the anæmia of insidious acute rheumatism, with heart lesion, for chlorosis with but a temporary relaxation and dilatation of the structures about the orifice.

Hypoplasia of the blood-vessels has been discussed already, and for a fuller account of these phenomena the reader is referred to the articles hereafter on "Congenital Malformation of the Heart" and "Diseases of the Arteries."

Thrombosis.—A remarkable and painful feature of some cases of chlorosis, happily rare, is the tendency of the blood to form thromboses in the cerebral sinuses, and indeed in other vessels of less immediate importance. Thrombosis may occur in such a vein as the femoral, or it may occur in the longitudinal or other cerebral sinus. Professor Osler quotes a case in which chlorotic thrombosis occurred in the axillary artery, with the consequent loss of the thumb and part of the fingers. The symptoms of thrombosis of the cerebral sinuses are dulness, stupor, vomiting, dilated pupils, delirium, and occasionally double choked disks. In a case under the late Dr. Bristowe tenderness and swelling of the right internal jugular vein appeared. This was followed by thrombosis in the right leg, yet the patient ultimately recovered. I have a vivid recollection of a similar case in the Leeds Infirmary in a servant girl of some twenty years of age. In other cases, of which I also remember one, hemiplegia may occur, and cases of this accident in chlorosis have been published by many observers. In thrombosis of the sinuses there is no palsy. Dr. Coupland says that this thrombosis does not occur in pernicious anæmia [art. "Pernicious Anæmia," p. 519]. Reference to cases of thrombosis will be found in the list at the end of this article, and in Professor Welch's article on "Thrombosis." Twice I have been much pained to hear of the sudden death from this accident of patients concerning whose ready recovery of health I had expressed myself confidently but a few days before.

Phlebitis, especially in the legs, is no very rare event in chlorotic women, and it is said to be more often bilateral in them (39, 43, 6, 41). It occurs in grave cases of chlorosis, and has been attributed to fatigue and chill. Its progress is usually rapid, and, accidents apart, the prognosis is very favourable.

Genito-urinary system.—In some cases of chlorosis the pelvic organs, like the arterial, are found ill-developed—the uterine hypoplasia of Virchow. It is difficult to believe that these cases are ever cured by medical means, or by any means. They find their way into the museums of pathology. Amenorrhœa is of course a feature of them.

Amenorrhœa is also usual in ordinary chlorosis, though it is far from invariable. Amenorrhœa is not only the ordinary condition, but also the most advantageous; indeed, it may be called the protective side of the process. If I may speak from a few examinations, I would say that in the cases of chlorosis in which the red corpuscles are numerically much diminished (say to 3,000,000 or under) menorrhagia or even menstruation in normal quantity (which is a relative menorrhagia in such persons) is or recently has been present. I find that Sir R. Gowers has observed falls of 10 to 20 per cent in the number of the red corpuscles after a menstrual period. These cases are less easy to cure. I need not say that many chlorotic girls are brought to us in order that the menses may be recalled; and we have to explain to the friends that if, by local and specific means, such an achievement were possible, the step would be rather a misfortune than a blessing. As an old medical friend of mine used to say to troublesome mothers, "Madam, when the works are put in order the clock will strike." In chlorosis a very slight loss of blood will intensify the impoverishment of the blood beyond all expectation.

Chlorosis may appear before menstruation has ever shown itself. Stockman in 63 cases found menstruation scanty or irregular in 29, absent in 12, normal in 4, profuse in 10. Three girls (aged 13, 15, and 19) had never menstruated, and in five no note had been made (normal?). Leucorrhœa is not infrequently complained of, and is cured by the iron. Of the urine I have already spoken; it is as a rule poor rather than loaded by products of waste, whether normal or abnormal. In particular there is an absence of those elements, such as indican and the conjugate sulphates, which would signify excessive fermentation in the intestines and absorption of toxins into the circulation. In pernicious anæmia the urine, like the skin, is usually darkened by the presence of urobilin in excess.

Nervo-muscular system.—Girls and young men alike, as they are adolescent, often go through phases of temper which are a source of anxiety to their friends; more new impressions, more new desires crowd in upon them than they can set in due order and subordination. It will not do, then, to put down the caprices, passions, perversities, and apathies of this season of life to any one of its disorders. They may occur even in the healthiest of both sexes; and with a little patience and protection from folly will "defæcate to a pure transparency." Yet chlorosis has, no doubt, some fretfulness of its own; lassitude and irritability meet together, and are due to want of activity in the nervous centres. There is no staying power; and although there may be proteids enough for repair, a small quantity easily provided of an element in which chlorotic blood is not deficient, yet the oxidation of carbohydrates and hydrocarbons for the supply of energy is behindhand. Fatigue products, also unoxidised, accumulate in the muscles. Dr. Sansom is disposed to attribute the fatty and other degenerations of the heart to a direct influence from the nervous system; at any rate we see irritability and loss of control (inhibition) in those higher centres which are the last to develop and the first to feel the lack of good blood. With these perturbations neuralgias are common, especially the neuralgias of the face, and headaches—frontal, temporal, or vertical. Gastralgia and pain under the left breast are common troubles of the chlorotic; the latter often coexists with leucorrhœa and disorderly heart, and with hysteria. Dr. Head has shown that all referred pains, with their accompanying tenderness, are apt to spread widely under the influence of anæmia. Thus widespread "neuralgic" pain and superficial tenderness may, in anæmia, be due to some simple cause. In the same way the headaches so common in anæmia are, in the majority of cases, a true referred pain, accompanied by tenderness, and correlated with pain and tenderness of a like nature on the chest or abdomen, according to the laws he has laid down. In a certain number of cases the headache and neuralgia represent a widespread pain referred from some organ of the head, such as the eye or the teeth. In extremely few cases, apart from pernicious anæmia and its allies, is the headache directly originated by the anæmia, though its wide distribution and prominence as a symptom are due to this cause. Certain kinds of palsy have been indicated under the circulatory system.

Optic neuritis is discovered occasionally in chlorosis; but the nature of its association with this malady is wholly unknown. Sometimes it appears rapidly as a papillitis, as in many cases of tumour. The prognosis is probably favourable: I have never come across an instance of permanent injury to vision in this kind. The sign may, however, embarrass the diagnosis, especially if headache be present. Choked disk may be seen in cases of thrombosis.

Diagnosis.—The chief difficulty in the diagnosis is to distinguish chlorosis from other anæmias, simple or toxic. Gilbert lays much stress upon the doctrine that upon chlorosis this anæmia or that may be superposed, or that two kinds of anæmia may be associated from the first in one person. Dr. Lloyd Jones also points out the same difficulty. If this be so, and there is strong reason to suppose that thus it is, the diagnosis in a given case of chlorosis may be no easy matter. We have seen that chlorosis is more than a simple anæmia following with uniformity on the withdrawal of blood, and menstruation, unfavourable conditions of life or work, or lactation may " superpose " a simple anæmia on the chlorotic (chloro-anæmia). I have suggested that such may be the compound causation of those cases of chlorosis in which the number of red corpuscles is very deficient (3,000,000 and under). A further difficulty, and a far more important one, lies in the possible confusion between chlorosis and such toxic anæmias as plumbism, rheumatism, chronic Bright's disease, syphilis, arsenic poisoning, and so forth. Of all these puzzles we see striking examples. One young lady, with a green pale face and menorrhagia, presented on closer examination a blue line on the gums. In the drinking-water lead was found in considerable quantities. In another such case, one which resisted all treatment, after a protracted search for some external cause, we discovered arsenic in large quantity in the green unsized wall-wash of her own sitting-room (not a paper). On the removal of this wash the symptoms gradually subsided. Syphilis does not give us so much trouble in women as in men, but is not to be forgotten. The quick effects of its specific remedies may betray syphilitic anæmia and place the diagnosis beyond doubt. Rheumatism is often insidious in young people, it is a potent cause of anæmia, and its murmurs more than "dynamic." To the anæmia of malignant disease I need not refer; I do not remember any difficulty in such cases. But in chronic Bright's disease in young persons I have not infrequently felt a brief indecision. The touch of the pulse will in all probability put the observer on the right line, and an examination of the retina and of the urine should settle the diagnosis. It is stated by some authors, as I have said, that a pulse of high pressure is apt to arise in chlorosis; this may perhaps occur in constipated patients. However, an examination of the urine will rarely fail to indicate the correct diagnosis.

The anæmia which precedes the appearance of pulmonary phthisis may create embarrassment in some cases; the absence of murmurs may guide us more or less, and the thermometer may come to our assistance. Dyspepsia may accompany any anæmia; there is nothing characteristic in the dyspepsia of chlorosis; and if an organic murmur be also present we may find it impossible to arrive at a certain diagnosis without delay. Fagge published a case from the records of Guy's Hospital, in a girl of 18, in whose case the diagnosis of chlorosis was upset on the post-mortem table; a large caseous mass of tubercle was dislodged from the cerebellum, and a few scattered tubercles were found also in the lungs. The blood was not systematically examined in those days (1861). The specific bacilli cannot be found in such cases.

For the diagnosis between chlorosis and splenic anæmia, a disease of the "chlorotic type," the reader is referred to the following article on this latter subject.

Anchylostoma, we are told, produces a state not always to be distinguished from chlorosis, not even by examination of the blood. The blood in anchylostoma, however, is generally said to present the characters rather of pernicious anæmia than of chlorosis. Pernicious anæmia is more readily to be distinguished from the latter by the blood, with the aid, perhaps, of the thermometer, and of an examination of the urine (*vide* art. "Pernicious Anæmia," p. 519).

Addison's disease might give us pause for a while. I remember one such doubtful case in a young woman; but even in the absence of pigmentations a careful survey of the symptoms and history of the case should preserve us from error.

Prognosis.—Chlorosis has never seemed to me to be the obstinate disease that it is for some writers. I may have been fortunate in not meeting with bad cases of it; still, although my experience of chlorosis has chanced to be exceptionally large, I recall few cases which seriously resisted treatment. The danger is lest the disorder relapse time after time. How this is to be prevented we shall consider in the subsection on treatment.

Prof. Stockman tells us that of his 63 cases 27 were in the first attack, 11 in the second, and 22 had suffered from more than two attacks. Many of these, he adds, did not persist in the remedies ordered for them, and became chronically anæmic. Some persons relapse in spite of all care; their blood is perpetually unstable, and iron is a necessary aid even in middle and later life. Dr. Stockman estimates the time of apparent recovery at four to six weeks. This period will be considered more fully under the head of treatment.

I find myself at some disagreement with those who say that phthisis is to be feared as a sequel of chlorosis. It is impossible to be assured that a patient weakened by chlorosis, or by any other malady, will not fall into phthisis; yet in my experience this sequel, far from being a common result, is indeed somewhat rare. It may be that the "prephthisical anæmia" has been occasionally mistaken for chlorosis. Gastric ulcer is more to be feared, though the causes of its association with chlorosis lie in obscurity. Of apoplexy and thrombosis of the sinuses I have already spoken. Happily they are events too rare to enter into ordinary forecasts. I repeat that the thing to be feared is relapse after relapse of the chlorosis itself. No careful prognosis can be given without repeated examinations of the blood. It is more than possible that many cases of chlorosis recorded as aberrant or peculiar were not cases of chlorosis at all.

Treatment.—I may almost paraphrase the words of Professor Osler in respect of quinine and malarious fever: "The physician who cannot treat chlorosis successfully with iron should abandon the practice of medicine" (vol. ii. p. 742). Physicians who restlessly turn from one preparation of iron to another, and from one drug to another, in order to find a cure for unmanageable chlorosis, must meet with peculiar cases. It is only by a strong effort of memory that I can recollect any cases of chlorosis in persons of common sense and reasonable obedience in which iron failed to effect a cure. On the other hand, I have had many cases submitted to me as intractable in which, if time and opportunity were favourable, there was no difficulty in compassing a cure. How are we to explain the failures? The reasons may be two: first, that iron failed of success because given in insufficient quantity; and, secondly, that the treatment was not continued long enough to counteract the strong bent to relapse which is seen perhaps in all cases more or less, and in some most doggedly. This latter reason covers, I suspect, most cases of failure. It is well never to begin to treat a case of chlorosis without telling the patient that the first course of medicine will extend to no less than three months; and that for a year thereafter she must be re-examined, and in all probability submitted to further courses of ferruginous tonics, as the signs may indicate. In all cases of severity the blood should be examined regularly, and this process has the incidental advantage of keeping the importance of the matter before the patient's eyes. During the first two or three weeks of ferruginous treatment the red corpuscles will rise quickly to the normal standard in number; though not in size, colour, or vitality.

It has been said of late that the first change to be seen is an increase of white corpuscles, and that these bodies act in some way as carriers of iron to the red. The manifold conditions on which the increased apparition if not the increased generation of white corpuscles depends are so little understood that we cannot be sure when an afflux of them is other than incidental.

The numerical increase of red corpuscles gives rise to a sense of relief often so rapid and so great that the unwarned patient jumps to the conclusion that she is "all right again"; and may throw medicine to the dogs. If so, the case may well be an "incurable" one. The increase of hæmoglobin, and the attainment of full growth by the corpuscles—which are the essential elements in recovery as are the reverse processes in falling ill—take place much more slowly. It is no uncommon thing to find that a return of hæmoglobin to the normal standard takes as long as three months; and for this reason three months should be enjoined as the shortest time in which a cure is to be completed. Even then relapse is more common than not. When I began practice, iron was given in doses too small to effect a satisfactory amendment, and gradually it became apparent that larger doses are required. Now there is a reaction, and physicians are saying that smaller doses suffice. My own opinion is that in cases of any severity, if recovery is to be ensured, iron must be given with a liberal hand; the quantity of the metal is more important than the particular preparation. Without returning to what has been said concerning the mode of operation of iron in chlorosis, I may remind the reader that, although in anæmia of simpler kind, as for instance after a hæmorrhage, "food iron" is adequate to bring about a repair, the iron given in medicinal doses in chlorosis must certainly have some further effect than the mere replacement of that required to rebuild the hæmoglobin; it must have some stimulant, tonic, or "specific" action which conspires to the same end. A few grains of the ammonio-citrate of iron is not a dose to cure chlorosis of any severity; far more than this may be needed. It is my custom to use the sulphate of iron alone, or with aloes, in the form of pill. The addition of an alkali to the iron is quite useless, and by making the pills more bulky is inconvenient. I generally administer one grain of the sulphate thrice daily after meals for the first week, two grains in the second week, three grains in the third; it is rarely necessary to go beyond this, though some patients do not respond till five-grain doses are reached; this, however, is exceptional. When the dose of three grains is reached, I direct that this quantity—nine grains daily—shall be continued for two months; the dose is then reduced by a grain, and thus administered for a fortnight; then one-grain doses are ordered for a month. During this time the pulse is probably settling to the normal rate, and if for a month before the end of this course the hæmoglobin has been constant at the normal standard a relapse is not very likely to occur; though of course the disorder may reappear after a time from the original causes. Recently I came across some little lozenges containing iron, called "jelloids." These I have found very successful, partly no doubt because being convenient and palatable, and arousing no fears of injury to the teeth, they are taken regularly, partly because they retain their free solubility. Occasionally the sulphate of iron causes some gastric irritation, the "jelloids" seem not to do so. I have often suspected that incurable chlorosis may mean insoluble pills; pills made up, for instance, with gum tragacanth and the like become as hard as pebbles and about as useful to the patient. For the flushed chlorotic patients (p. 498) the laxative iron mixtures are indicated, such as the combinations of tincture of the perchloride with sulphate of magnesia; or of equal parts of Griffith's mixture and compound decoction of aloes, a most efficacious medicine and not so nasty as it looks.

It was an imposing lesson of our youth that iron is not to be given till the patient is "prepared" for it; and to this end bottlefuls of soda and gentian, and so forth, would be prescribed; far be it from me to encourage a careless mode of administering any drug, yet nevertheless I think this so-called preparation was otiose and even mischievous in so far as it wasted time. I rarely find such preparatory courses necessary. If the tongue be white and sticky and the bowels constipated let a blue pill and a dose of salts be given; this done, begin with the iron, and watch the remainder of the tongue-cleaning process going on fast enough under the iron. The dyspepsia being in most instances the consequence of the deprivation of oxygen, the assimilative changes will improve, without any direct attention, as the hæmoglobin is restored. In exceptional cases, no doubt, some precautionary measures may be desirable; of these the physician will judge.

I have tried all or most of the so-called preparations of "organic iron" produced for us by our excellent allies the manufacturing druggists; helpful as many of their novelties are, I regret to say that "organic iron" does not seem to be one of them. Perhaps I may make an exception in favour of an old-fashioned French solution of malate of iron which I have found that patients with queasy stomachs can take when ordinary ferruginous drugs are ill borne or seem inappropriate. Gilbert has found the protoxalate very useful; it is said to be soluble in the gastric juice. Stockman indeed says that "inorganic iron" is more rapidly effective than "organic iron."

Of "adjuvants" teachers and friends recommend many to us; ether, liquor ammoniæ acetatis, nux vomica, and so forth: but I cannot say that I have found in any of them more advantage than such as may flow in the individual case from the ordinary properties of these accessories; they may be needed or they may not, usually not. It is well, however, to add some cordial such as chloric ether or sal volatile to all steel mixtures.

On pathological grounds much has been made of late of an antiseptic treatment. In a paper lately read at Cambridge by Dr. Latham, great stress was laid by the author on the value of the liquor of the perchloride of iron, because, as he showed at the time, it contains much free chlorine. Dr. Latham's claims on behalf of this vehicle of iron are probably well founded, as they are in accord with other observations of the kind. For instance, Townsend thus tabulated his results in 87 cases:—

	Hgbn. incr.
β-naphthol (30 cases)	1·85 per cent
Blaud's pills (31 cases)	5·07 „
Naphthol first and afterwards Blaud's pills (12 cases) .	6·70 „
Blaud's pills alone (19 cases)	4·50 „

This table shows a decided advantage in favour of the use of β-naphthol before the pills; and in another such series, of 28 cases, the Hgbn. increase was 7·9 per cent. In the Boston Medical Society, to which body this paper was read, it was generally agreed that intestinal antisepsis combined with iron gives better results than iron alone.

I remember in a few cases, when for some reason the iron did not take good hold at first, the drug seemed to get a start on the addition of arsenic or phosphide of zinc; ordinarily to treat chlorosis with these drugs is, to say the least, a waste of time.

In the belief that in chlorosis the volume of the serum is increased (serous plethora), bleeding and diaphoresis have been recommended as means of cure. It is not apparent how an operation which reduces the number of the red corpuscles can be otherwise than injurious. However, Schmidt treated and tabulated the following eight cases (a "bleeding" was 80 ccm.) :—

	Average incr. Hbgn. per cent.	Weekly incr. of weight in kilos.
i. One bleeding and iron	6·20	0·73
ii. Iron alone	6·18	0·48
iii. One bleeding	2·50	0·92
iv. Several bleedings	0·59	0·51
v. Sweating cure	0·39	0·44
vi. Bleeding and sweating	0·36	0·46
vii. Several bleedings, sweating, and iron	0·02	0·04
viii. Several bleedings and sweatings; no iron	0·56	0·19

The good effect in the first case was entirely due, no doubt, to the iron.

While prescribing pharmaceutical remedies the physician will not forget, so far as in him lies, to rectify such disadvantages of life as he may be able to ascertain. Over-pressure at school, unwholesome conditions of work or amusement, late hours, worry, tight lacing are points to which his attention will be directed; yet while relaxing overwork, if any, he will be no less alive to the evil of idleness or desultoriness. As much time as possible should be spent in the open air and in such gentle exercise as the strength and respiratory functions will permit. Quiet horse exercise or cycling may be encouraged, and some course of study likewise which shall interest and discipline the mind and temper without fatigue. The patient should sleep, if possible, with the bedroom window open; if this be prevented by hard weather, the door must be open. A cold bath will probably prove more than the deficient heat production can support, but the rapid application of the wet sheet can usually be prescribed with advantage; this is better done in the forenoon two hours after breakfast, and, during the colder months, in a room with a fire. Excessive cold, as we see in hæmoglobinuria, seems to destroy the red corpuscles. If love affairs harass the patient it must be remembered that marriage is no direct cure.

I have said that the deficient powers of heat production often forbid too bracing a line of treatment; in a bad case of chlorosis, one in which perhaps iron is not telling at once, the dissipation of heat and the expense of muscular activity must be husbanded by a week or a fortnight in bed. Such a measure often gives an impulse to the curative movement, and proves to be an economy of time in the end. The facial, gastric, and other neuralgias, which may be prominent symptoms of the case, are usually relieved at once by this simple means. A further reason for recumbency is given by Dr. George Oliver in his interesting little book on *Pulse-Gauging.*

Thus the calibre of the arteries is enlarged, residual blood in the ventricles is reduced, and dilatation of the heart is prevented or relieved. By his arteriometer Oliver finds (p. 135) "that from 25 to 100 per cent more blood is discharged into the tissues in the recumbent than obtains in the sitting posture . . . the radial calibre . . . increases in recumbency, as a rule, in proportion to the severity of the anæmia and to the need of recumbent rest." As soon as the appetite improves and the other graver symptoms begin to give way, change to the seaside or to the hills may be advised; but cold, I repeat, is injurious in chlorosis, and at considerable altitudes the deficiency of oxygen would be more and more sensibly felt.

It only remains now to say the few words which are necessary on the diet of chlorosis. It is of the first importance to overcome the common distaste for meat. Girls will say that the entry of a dish of hot meat into the room makes them feel sick; kindly and gradually this aversion must be overcome, and meat must take its due place in the diet. Eggs and milk if well digested will be included, and sweets and other kickshaws discouraged. Green vegetables are said to be useful for their chlorophyll, at any rate they avert constipation. It is desirable, if a fair meal be taken, that nothing be offered between meals. We are pointedly asked in these cases of chlorosis whether alcohol in any form is to be prescribed. Of itself I believe that alcohol is of no direct service. It is possible now and then that a bad appetite may be coaxed into more activity by a glass of stout, or of red wine and water; if so, the use of these aids is justified. Some young persons dislike pure water; and indeed it is not well for these chlorotics to drink much with meals: half a tumbler of milk may be the table drink, and three hours after meals a glass of hot water will act beneficially both on the stomach and on the secretions. Careful mastication of the food is of great importance.

In conclusion I would repeat that to test the blood not only for the number of red corpuscles and apparent hæmoglobin value, but also to ascertain whether they are equal and of full size, is the only trustworthy means of gauging the rate and degree of cure; a lowered pulse-rate, however, is a sign of amendment, as is reacceleration of impending relapse. Colour generally returns to the face and steadiness to the breathing long before the cure is established.

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PERNICIOUS ANÆMIA

SYNONYMS.—*Idiopathic anæmia; Essential anæmia; Myelogenic anæmia; Progressive pernicious anæmia; Ganglionic anæmia; Anæmatosis.*

Definition.—By "pernicious anæmia" is now generally understood a variety of primary anæmia, which arises insidiously, and is characterised by progressive diminution in the number and changes in the form of the red corpuscles of the blood, together with a similar but generally less marked diminution in the amount of hæmoglobin; which changes apparently depend upon undue hæmolysis combined with inadequate compensatory hæmogenesis,—a condition which, in the majority of cases, passes more or less rapidly to a fatal termination, the progress being in some cases interrupted by periods of improvement followed by relapse, but rarely resulting in permanent restoration to health, whatever the method of treatment.

It is difficult, even in the light of modern research, to frame a satisfactory definition of this affection—one which shall not be too wide, nor, on the other hand, too narrow to embrace the varied conditions under which this severe form of anæmia is known to arise. There is reason to doubt even the constancy of the "progressive" and "pernicious" characters which were deemed by Biermer to constitute its most characteristic features. On the other hand, the use of such names as "idiopathic" and "essential" anæmia, however appropriate they may have been when Addison directed attention to this class of primary blood affection, can hardly be justified now except as an admission that pathological research has failed to discover the source of a disease which presents such striking clinical features. The difficulty is enhanced by the fact that the clinical phenomena are not in themselves distinctive, not even the characters of the blood; hence, as the advance of knowledge led to the shrinking of the "idiopathic" area, it seemed reasonable at one time to admit the existence of secondary forms of pernicious anæmia side by side with the primary.

However, there is good ground for believing that when all extrinsic causes are eliminated, there remains a residue of cases of progressive anæmia, to which the term "primary" may be assigned; and it is to this class that we may also assign, at least provisionally, the name "pernicious." For a primary anæmia may be defined as one dependent on the perverted relationship between the two great functions concerned in maintaining the normal composition of the blood. In the active processes of disintegration and renewal of the blood elements, hæmolysis is balanced by hæmogenesis. The balance may be disturbed by excessive hæmolysis on the one hand, or by inadequate hæmogenesis on the other.

It will be seen that an explanation of idiopathic anæmia has been sought in each of these directions severally, but, as stated in the above definition, there may be defects in both. Whether the definition should be made more precise by indicating the probable source and character of the hæmolytic process is a matter which I shall discuss under the head of "Pathology."

History.—By common consent the credit of the first general account of pernicious anæmia is due to Dr. Thomas Addison, whose reference to it in his monograph "On the Constitutional and Local Effects of Disease of the Suprarenal Capsules" has often been quoted. Although these passages appeared in 1855 he had been teaching their doctrines for several years. No account of the condition can be considered complete without Addison's description of it:—

"For a long period I had from time to time met with a very remarkable form of general anæmia occurring without any discoverable cause whatever, cases in which there had been no previous loss of blood, no exhausting diarrhœa, no chlorosis, no purpura, no renal, splenic, miasmatic, glandular, strumous, or malignant disease.

"Accordingly, in speaking of this form in clinical lectures, I, perhaps with little propriety, applied to it the term 'idiopathic' to distinguish it from cases in which there existed more or less evidence of some of the usual causes or concomitants of the anæmic state.

"The disease presented in every instance the same general character, and, with scarcely a single exception, was followed after a variable period by the same result.

"It occurs in both sexes; generally, but not exclusively, beyond the middle period of life; and, so far as I at present know, chiefly in persons of a somewhat large and bulky frame, and with a strongly-marked tendency to the formation of fat.

"It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to his earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted; the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless; the flabbiness of the solids increases; the appetite fails; extreme languor and faintness supervene, breathlessness and palpitations being produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles. The debility becomes extreme; the patient can no longer rise from his bed; the mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires. Nevertheless to the very last, and after a sickness of perhaps several months' duration, the bulkiness of the general frame and the obesity often present a most striking contrast to the failure and exhaustion observable in every other respect.

"With perhaps a single exception, the disease, in my own experience, resisted all remedial efforts, and sooner or later terminated fatally.

"On examining the bodies of such patients after death I have failed to discover any organic lesion that could properly or reasonably be assigned as an adequate cause of such serious consequences; nevertheless, from the disease having uniformly occurred in fat people, I was naturally led to entertain a suspicion that some form of fatty degeneration might have a share at least in its production; and I may observe that, in the case last examined, the heart had undergone such a change, and that a portion of the semilunar ganglion and solar plexus, on being subjected to microscopic examination, was pronounced by Mr. Quekett to have passed into a corresponding condition.

"Whether any or all of these morbid changes are essentially concerned—as I believe they are—in giving rise to this very remarkable disease, future observation will probably decide."

Isolated examples of the remarkable condition thus succinctly described by Addison had found their way into medical records prior to the date of his writing. This has been shown by Lépine and Pye-Smith in their historical summaries of the subject. Thus, the latter author refers to the records of seven cases which doubtless fall into the category of "Addison's anæmia"; namely, one recorded by Combe (1823), one by Andral (1823), one by Marshall Hall (1837), one by Piorry (1841), one by Pearce (1845), and two by Barclay (1851). It seems clear, too, that the cases given by Channing of Boston (Mass.), in a paper, written in 1842, dealing with anæmia in relation to the puerperal state and in uterine disorders, probably belong to the same class. However, comparatively little attention was attracted to the subject for several years after Addison wrote, except, it is right to add, amongst those who were most familiar with his teaching. Thus, Sir S. Wilks, in the Guy's Hospital Reports for 1857, discussed idiopathic fatty degeneration, in which he referred to the morbid change which is the most characteristic feature of idiopathic anæmia; and cases were recorded by Habershon and others of the Guy's school. The writings of Gusserow and Biermer of Zurich, especially the memoir in which the latter author first uses the phrase "progressive pernicious anæmia" (1871-72), did much to awaken interest in the subject, and, I may add, to ignore the previous work of Addison; thus to Biermer was given the credit, which indeed he himself claimed, of describing for the first time the characters of a condition not hitherto recognised. It is clear, however, that not only had he been anticipated by Addison, but it is also probable that the cases amongst pregnant women to which Gusserow drew attention were the same as those indicated by Channing thirty years previously, and by Lebert in a case recorded in 1853 as "puerperal chlorosis." Nevertheless, the service rendered by Biermer was considerable, both from the clinical and pathological standpoint; and his choice of the name "pernicious" directed attention to the fatal character of the disease. However, we must recognise that, as knowledge has grown, reasons have increased for believing that many cases included by Addison as "idiopathic" or by Biermer as "pernicious" would more strictly find a place amongst the secondary anæmias. For not only have unsuspected causes been revealed of progressively fatal anæmias which exhibit all the characters described by Addison and Biermer (such as anchylostomiasis), but definite, if minute, pathological differences have been shown to exist between pernicious anæmia proper and certain other cases which some years ago I regarded as examples of "symptomatic (secondary) pernicious" anæmia; for in the predominance of the cardinal symptoms masking the underlying condition, these latter cases ran a course and exhibited many characters which resembled those of cases not so associated, and therefore named provisionally "idiopathic." This is not a mere question of nomenclature, it concerns the true interpretation of the pathology of the disease before us, and must be referred to again when we speak of this branch of the subject. We must constantly bear in mind, therefore, that a certain number of the cases on record are not strictly to be ranked in the category of "pernicious anæmia" properly so called.

It is not possible in such an article as this to review all that has been written during the past twenty-five years upon the subject. Many contributions have been made by physicians of Addison's own hospital, namely, by Pye-Smith, F. Taylor, and Hale White. Most of their writings appear in the Guy's Hospital Reports. Dr. Stephen Mackenzie, again, in a clinical lecture published in 1878, did much to invite attention to a subject to which he reverted some years later in the Lettsomian lectures which he delivered in 1891 at the Medical Society of London. Dr. Byrom Bramwell published a full account of pernicious anæmia early in 1876, and drew attention to the value of arsenic in its treatment. To Dr. W. Hunter we owe some of the most profound studies of the pathology of the disease, studies which have materially influenced our conceptions of its nature, and have done more than any other work to give definiteness to them. Other labourers in the same field have been W. Russell, Brakenridge, Gibson, Stockman, and Fraser of Edinburgh; Finny, Purser and Craig of Dublin; Mott, Russell, and James Taylor of London. The disease was studied at an early date in America, the contributions of Osler, Gardiner, and Howard of Montreal being amongst the first; Osler is also the author of many subsequent studies. To Pepper of Philadelphia is due the discovery of changes in the marrow of the bones, observations speedily confirmed by others. Musser, Henry, Kinnicutt, and Woods have also contributed to the subject in the United States. Numerous essays and monographs have appeared in the Continental schools by Eichhorst, Quincke, Muller, Neumann, Immermann, Lépine, Hayem, Laache, and others.

Etiology.—To speak of the causation of a disease of which, in the majority of cases, no adequate cause can be discovered seems paradoxical;

yet in pernicious anæmia we detect certain remoter antecedents, which, if not of themselves adequate to give rise to the condition, nor constant in their occurrence, are yet not without importance. These are among the more general causes of anæmia, for anæmia owns an infinity of causes; but they seem to have no bearing on the quality of perniciousness. Whenever the anæmia appears to pass beyond the boundary of the incidental, and becomes so dominating a feature of the illness that, as it increases, all the symptoms are referable to the anæmia, and none to the primary affection, then it is legitimate to infer the intervention of some further agency which has converted the simple into the pernicious type of anæmia. Thus, for example, hæmorrhage is a common cause of secondary anæmia, and repeated small bleedings may produce a condition which progresses from bad to worse, and ultimately destroys life. Is such a state to be regarded as "pernicious anæmia"? Some have thought so, and a recent writer (Stockman) has striven to show that the very hæmorrhagic tendency which belongs to severe anæmia may be the means of its passing by a vicious circle into a pernicious and fatal disease. Such a view, of course, gets rid of the notion that pernicious anæmia is a specific disease; as, thus, any anæmia of sufficiently high degree provides for its own further progress by the effects which it produces on the nutrition of the blood-vessels. On the other hand, that cases of hæmorrhage and of diseases involving large hæmorrhages, or a continued repetition of smaller ones, although leading to a chronic anæmia, do very rarely assume the type under discussion, seems to prove the existence of other determining factors; for it does certainly happen that, although a case of pernicious anæmia may seem to have been initiated by a metrorrhagia or a gastric hæmorrhage, the anæmia advances without any repetition of the loss of blood, whether from the primary source or from secondary sources opened by the anæmic state. Thus, even were there no other distinguishing characters—such, for example, as those of the blood and urine—we should have to assume the presence of some fresh factor to account for the course of the disease. The consideration of some of the more common antecedents of pernicious anæmia may suggest to us where this *tertium quid* is to be sought.

Amongst the favouring conditions upon which some stress has been laid are insufficiency or unsuitability of food in persons subjected to hard manual labour, or even not so subjected. The conditions of life of the Swiss peasantry were at one time supposed to determine the many instances of the affection which rendered Zurich a centre for its study. Misery and famine are conditions of anæmia; but unhappily such conditions are as common in the large centres of population as they are in the rural districts, yet pernicious anæmia is a rare disease by no means limited to the poorer classes of the community. Moreover, it is mainly in the writings of the Swiss authors that any reference is made to such etiological factors. Another class of antecedents most prominently cited in the writings of Gusserow and Biermer, to account for the excess of women among their patients, consists of pregnancy, parturition, and lactation; yet how seldom is this fatal kind of anæmia observed as a direct sequence of these physiological states. Reference has already been made to hæmorrhage as an antecedent, and mention must be made of gastro-intestinal disturbance also, which of all factors seems likely to be the most nearly connected with the etiology of the disease. Not only are the earliest symptoms connected with this system in a considerable proportion of cases, such as vomiting, diarrhœa, or irregularities of digestion, but many cases exhibit also definite changes in the gastric or intestinal mucosa; and this to such an extent as to have led some observers to attribute the fatal anæmia to the atrophy or other lesions which attack nutrition at its source (Fenwick, Kinnicutt, Osler). Again, it is well known that patients with chronic gastric disorders, such as ulcer or cancer, are sometimes anæmic out of all proportion to the amount of hæmorrhage which may have occurred during their illness. Indeed, in some cases where there are no direct symptoms of the gastric disorder the resemblance to pernicious anæmia is striking; and cases have been described where anæmia apparently pernicious has seemed to establish itself upon the gastric disease (Eisenlohr). The arguments of Hunter on this point have much force, his contention being that in all these conditions where the clinical features of pernicious anæmia are manifested in association with malignant disease or gastric changes, the lesions essential to the former have been superadded; for, as he points out, pernicious anæmia is a rare complication in malignant disease, whilst the gastric lesions often occur apart from pernicious anæmia. In commenting on the view, advanced by Fenwick, that atrophy of the gastric glands underlies pernicious anæmia, Hunter points out that this observer himself found such atrophy in a large number of cases of cancer also. "Thus, of fifteen cases of cancer of the breast, in only four were no anatomical changes to be found in the gastric mucosa. Some degree of atrophy was found in every case of cancer of the stomach. If atrophy of the gastric glands is to be regarded as the essential anatomical change in pernicious anæmia, it would seem reasonable to expect that pernicious anæmia should be found frequently associated with cancer of the breast, and almost invariably with cancer of the stomach. Curiously enough, however, I have not found a single case recorded in which cancer of the breast has presented the features of pernicious anæmia; and as regards cancer of the stomach, it is the exception and not the rule for it to be marked by the clinical features characteristic of pernicious anæmia" (Hunter). Similar reasoning is employed by Hunter to cast doubt on the alleged etiological importance of the degenerative changes described by Sasaki and by Banti in the nervous tissues of the intestinal wall; for such changes, according to the observations of Scheimpflug, are frequent in wasting and acute infectious diseases. Banti, regarding the sympathetic nerve lesions as a primary defect, went so far as to give the name "ganglionic anæmia" to this affection.

Among the influences which have been known to precede a progressive anæmia must be included those in which the mental and emotional faculties are concerned. There is authentic evidence that shock, depression, anxiety, or severe mental strain have been followed by the appearance of an anæmia which has run on to a fatal issue. The precise relationship between the disease and such antecedents is, however, quite obscure.

The hypothesis that pernicious anæmia is due to microbic agencies has little to support it; the micro-organisms that have been described in the blood (Fränkenhauser) have not been isolated or cultivated, whilst the condition of the blood itself is such as readily to lead to errors of observation in this respect.

Lastly, pernicious anæmia is mainly a disease of adult life, most cases occurring between the ages of twenty and forty-five. But children are not exempt from it, cases being recorded in patients as young as seven, eleven, and twelve years. The sexes are about equally prone to it, but if all cases of its apparent origin in pregnancy and parturition be excluded, it would probably be found that the disease preponderates amongst men.

Symptoms.—In the vast majority of cases it is extremely difficult to fix the date at which the illness began; its onset being so gradual and insidious that the patient passed imperceptibly from health to disease. It does, however, occasionally happen that a debilitating illness, a great loss of blood, pregnancy, or parturition has been followed immediately by an anæmia of the ingravescent course characteristic of the disease; and, as has already been pointed out, even some unusual mental shock or emotional disturbance may be directly antecedent to the appearance of the anæmia. Whether or not we are to include in the present category every case which seems to have its origin so directly in an anæmiating cause, it is evident nevertheless that the declared symptoms do not differ in kind from those which are thus produced. There is hardly a single symptom of the protopathic affection which is not to be found now and again in association with profound anæmia clearly due to an ordinary cause. The earliest indications of the malady are so slight and insignificant as to be disregarded, and it is often not until the disease is well advanced that its true nature is recognised. Nor is it possible to assign any period during which these early and indefinite signs may be said to last. It is a stage measured mostly by weeks or even months rather than days, although cases of apparently acute course are on record. These *initial symptoms* consist in the main in failing strength, and in disinclination for exertion, physical or mental; so that the subject of the malady becomes possessed by an unnatural lassitude which makes all labour irksome, and often renders him despondent, low-spirited, and capricious in temper. Together with this persistent asthenia and loss of energy the appetite fails; there may be complaint of discomfort after food, and the patient will perhaps suffer at times from nausea or even vomiting. If to these gastric disturbances there be added intestinal irregularity, it is natural for him and his friends to ascribe his weakness and depression to some functional derangement of the digestive organs. Yet, as a rule, there is no falling off in nutrition, but even a noticeable increase in bulk and weight. Gradually, however, the signs of anæmia are more evident, and, as they become pronounced, his weakness increases, he suffers from palpitation, perhaps from syncopal attacks, and shortness of breath on exertion, and at last is compelled to abandon his calling and seek rest and advice. The symptoms which may now be presented, those, that is, of the declared disease, may best be described in detail; they occur with variable frequency, and some even which may be thought to be essential and characteristic are occasionally conspicuous by their absence.

The constant symptom is of course *the anæmia.* The pallor of the skin is striking, often in marked contrast with the previous good colour of the individual. The skin and mucous membranes are almost devoid of colour, save that the former, especially of the face, generally assumes a faint yellowish or lemon tint that is wholly different from the whiteness of the subject of pulmonary tuberculosis, the earthy pallor of the cancerous cachexia, or the muddy tint of the malarial subject; and is quite different from the bronzing of the malady discovered by Addison in his search for an adequate cause of "idiopathic" anæmia. This complexion, however, is sometimes met with in the chlorotic, in those who have suffered from internal hæmorrhage (as, for instance, in cases of large pelvic hæmatocele in process of absorption), in rare cases of chronic gastric organic disease, and in various toxic anæmias. It cannot be deemed pathognomonic; but occurring, as it does, with so few symptoms, or overt evidence of blood loss, it may lead to the suspicion of the grave nature of the malady. It is all the more suggestive when it occurs in a male subject of mature age whose previous health record has been excellent. There may be some œdema of the lower extremities, often very slight, sufficient to cause slight pitting on pressure over the malleoli; sometimes more extensive, and not seldom entirely absent. Indeed, this symptom is hardly so frequent a feature of pernicious anæmia as it is of chlorosis. In the later stages, however, it may become marked, as also may petechial hæmorrhages chiefly on the lower limbs. There is no constant condition of skin as regards perspiration. Some have noticed undue sweating in the earlier and later periods of illness, but there does not appear to be any regularity in this symptom, and it can hardly be regarded as essential. The skin often assumes the soft and smooth character to be met with in the subject of fatty degeneration.

The temperature of the body is generally normal, and in advanced stages subnormal; but most observers record periods of remarkable pyrexial exacerbation, which some consider to be peculiar to this kind of anæmia. This pyrexia is not as a rule severe, the temperature seldom exceeds 102° or 103°, with morning remissions and marked irregularity. It may last for a few days and then subside, recurring at intervals during the progress of the malady; or it may be of more continued course. This intermittent pyrexia is possibly related to the variations in the hæmolytic process, and may be taken as confirmatory of the toxæmic theory of the disease; another conceivable view is that it is due to capillary hæmorrhages in the heat-controlling centres of the brain. Whatever the explanation it is seldom entirely absent; but then, it may be remarked, a subfebrile temperature is not uncommon in chlorosis, and may also occur after severe hæmorrhage.

The signs of *circulatory disturbance* are generally obvious, and may even lead to an erroneous opinion. The patient may complain of palpitation from an early period, and at times may be attacked with faintness or actual syncope. The pulse, generally quicker than normal, is markedly affected by exertion or emotion, the difference between its rates as influenced by posture being considerable. It is mostly soft and fairly full, whilst there may be complaint of throbbing in the neck or a feeling of fulness in the head. The cardio-vascular signs of anæmia are pronounced. The impulse of the heart is undulating, and the apex-beat generally somewhat lower and situated more to the left than normal; percussion confirms this evidence of slight dilatation of the ventricles by revealing an increase of the cardiac area of dulness to the left. An intense blowing systolic murmur is generally audible over the præcordium, most marked at the pulmonary cartilage. In advanced cases this murmur may have a grating character, and be even mistaken for friction. (A diastolic murmur has been noted, but this is not common.) Its hæmic origin is confirmed by the presence of a similar murmur in the large arteries, and a loud hum in the jugular vein. The carotids often pulsate violently, and a distinct thrill is to be felt over them and the large veins in the neck.

Examination of the blood reveals a great departure from the normal. The bloodless condition of the skin makes it somewhat difficult to obtain sufficient for its estimation. The drop has a pale watery appearance, and the number of red corpuscles is found to be notably diminished. The degree of this oligocythæmia depends upon the stage and severity of the disease; but it is not unusual to find the number of red corpuscles reduced to 1,000,000 per cub. mm., that is, 20 per cent of the normal; and as the case progresses they may fall considerably below this figure, the lowest estimation on record being 143,000 (Quincke). There is no parallel diminution in the number of leucocytes; in late stages they may exceed the normal amount. With the hæmoglobinometer it will almost invariably be found that although there is a marked reduction in the amount of hæmoglobin this is not proportionate to the reduction in corpuscular richness. Thus in a case in which the corpuscles have fallen to 10 per cent the hæmoglobin percentage may be as high as 20 or 25 per cent. Hence it follows that the individual corpuscles must have a larger relative content of hæmoglobin than in health. The microscopical examination shows notable changes, which when first observed were thought to be quite characteristic. In the first place, the tendency for the corpuscles to form rouleaux is almost entirely lost, although this is not to be observed in every case. As a rule the scanty corpuscles either form irregular masses, or lie scattered over the field; and it is evident at once that they exhibit very great variations in size and shape. Thus many are of irregular form, pear-shaped, oval, and deformed, constituting the condition named by Quincke "poikilocytosis," which, however, is not distinctive. Some are much larger than normal (megalocytes), and many, on the other hand, appear as small spherical bodies (microcytes). Neither of these varieties can be considered distinctive, although some authors think that the prevalence of the megalocytes is greater than in any other condition; others, however, consider that the microcytes are if anything more characteristic. Besides these forms, which may be regarded respectively as immature or overgrown red corpuscles, there are also to be seen nucleated corpuscles, which suggest a reversion to the reptilian type; these, according to their sizes, have been named normoblasts, megaloblasts, and microblasts. Lastly, Ehrlich showed that granular corpuscles which stain with reagents are often present, and are possibly degenerate corpuscles; whilst the readiness with which the hæmoglobin accumulates in a mass within a corpuscle gives the latter a pseudo-nucleated aspect. On the other hand, Dr. Copeman found that hæmoglobin separated from the corpuscles in pernicious anæmia with abnormal readiness. There is no increase in the number of leucocytes; but it has been observed that these are mostly of the smaller (lymphocyte) variety, and granular masses are present. Thus the changes are mainly limited to the red corpuscles, which may be considered to exhibit disintegrating forms and immature corpuscles side by side.

A *tendency to hæmorrhage* is a noteworthy feature of the disease. This may be seen comparatively early in its course, and may aggravate the anæmia; but in extent and degree it is too variable to be regarded as the direct cause of the progressive character of the affection. The hæmorrhages may take the form of epistaxis, of hæmatemesis, of hæmaturia, or of bleeding into internal organs, such as the brain and the spinal cord. Most commonly, however, they are but small capillary effusions, and occur most distinctly in the retina. It has been shown by Mackenzie that these retinal hæmorrhages are prone to occur in any form of prolonged anæmia if of sufficient intensity (below 50 per cent corpuscular richness); and as this degree of intensity is generally attained in pernicious anæmia their occurrence in this affection is one of its most common symptoms. The retinal hæmorrhage was first pointed out by Biermer, and may be regarded as a sign of some value. As a rule it does not lead to any impairment of vision, but cases have occurred where it has been the cause of amaurosis. Mention has already been made of the cutaneous hæmorrhages which may occur.

Dyspnœa is a prominent symptom, the enfeeblement of the heart adding to the respiratory difficulty caused by the lack of hæmoglobin. As the end is approached this symptom may become more marked and distressing, and the physical signs of œdema of lung may supervene. Otherwise there is comparative freedom from pulmonary disease throughout, although bronchitis and pneumonia have been observed as serious complications.

Disturbances of the gastro-intestinal system are among the earliest and most frequent features of the attack. The pale, flabby tongue denotes the general anæmia and want of tone of the stomach—conditions indicated by nausea, vomiting, epigastric uneasiness, and flatulence. The secretion is deficient in hydrochloric acid, in many cases it may be actually wanting (achlorhydria). Digestion is therefore slow, and the appetite much impaired. Sometimes irregularity of the bowels is marked by diarrhœa alternating with constipation, the former being occasionally profuse. So common and so early is the appearance of such digestive disorders as to give ground for the belief that they have an important influence in inducing the anæmic condition; and the fact that anatomical changes are frequently found in the stomach of subjects of pernicious anæmia has led some observers to ascribe the affection to atrophy of the gastric glands. Jaundice is not common, and when present seldom intense. Palpation reveals enlargement of the liver, with some tenderness over it. The spleen is rarely to be felt. Ascites may be present, but never in large amount.

The urine is generally fairly abundant, of normal or diminished specific gravity, acid, and free from deposit. It is often pale, but even in the course of the illness it may assume a high colour hardly consonant with the anæmic state. The chief cause of this coloration was shown by Hunter to be due to an excess of pathological urobilin, and the significance of this ingredient is considerable.[1] There may be an excess of indican, and free iron has been observed by some (Finny). Uric acid is generally in excess, but there is no constant change in the amount of urea. Occasionally albuminuria is noted, but it is not a prominent feature. Observers have noted the presence of many another abnormal constituent; according to Hunter, the presence of pathological urobilin, renal epithelium, casts containing blood pigment, and increased excretion of iron, is characteristic. There may be hæmaturia.

The symptoms exhibited on the side of the *nervous system* have received especial attention of late years since the discovery of definite organic change in the spinal cord in cases of profound anæmia. The functional disorders comprise irritability, a growing inability to fix attention upon a subject, loss of memory, and often marked insomnia. Headache is not prominent until the anæmia becomes extreme, when there may be delirium also, or even more violent mental disturbance. In some cases the end is ushered in by convulsions passing into coma; in others a lethargy gradually deepens into coma. But the intellect often remains unimpaired almost to the close, and death takes place from mere exhaustion. The occurrence of convulsions and the appearance of partial or complete hemiplegia, or monoplegia, denotes cerebral hæmorrhage of greater or less extent. Sometimes the paralysis is so slight and transient as to indicate that the hæmorrhage must have been very small; in others a definite apoplectic seizure terminates the illness. The spinal symptoms referred to above consist, in the main, in slight disorders of motility, mostly ataxic in character, sometimes spastic; and they have been found associated with pronounced changes in the spinal cord. At the same time some of these degenerative lesions have been met with in cases that did not exhibit any disorder of function during life.

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[1] Gowlland Hopkins, from examination of five cases, was unable to satisfy himself of the presence of "pathological" urobilin, and was inclined to attribute the spectroscopic indications to an admixture of urobilin and hæmatoporphyrin.

Morbid anatomy.—At the time of death the body appears, in the majority of cases, to be fairly well nourished. The pallor of the surface is striking, and petechiæ may be distributed over the lower extremities, which may be somewhat œdematous. The panniculus adiposus is often of a deep yellow colour, and the dark red tint of the muscular layers contrasts with the exsanguine-aspect of the skin. Some thin serous effusion may be found in the peritoneal and other serous sacs. The blood is thin and watery, and the clots in the cardiac cavities small and pale. The blood serum has been observed to have a yellowish tint from admixture with hæmoglobin readily liberated from the corpuscles, and even to stain the hands of the pathologist. The specific gravity of the blood is lower than normal; in one case it is stated to have been 1028 instead of about 1055. The microscopical characters of the blood have already been given. The heart is generally well covered by epicardial fat, and sometimes petechial hæmorrhages may be seen on its surface. The muscular substance is soft, flaccid, and of a tawny, brownish tint, sometimes compared to that of a faded leaf. The musculi papillares, especially of the left ventricle, are nearly always variegated by wavy whitish streaks—the "tabby-cat striation" of Quain. Microscopically the fibres are found to be in various stages of fatty degeneration, some wholly converted into fatty granular and oily detritus, others with accumulated fat granules around the muscle nuclei. The valves are normal, but small areas of opaque white fatty degeneration may stud the intima of the aorta. Similar fatty degeneration has been found in the arterioles and capillaries, leading often to their rupture in various parts of the body. The lungs present no notable lesion; they are, as are all the viscera, very bloodless; although mostly the lower lobes present some engorgement and œdema, and there may be petechiæ beneath the pleura. Occasionally it happens that the disease attacks a subject who presents some old caseous or cretified tubercle in the lung, and sometimes also an intercurrent pneumonia terminates life; but of course neither the old nor the recent changes are essential. The stomach exhibits a striking pallor of its mucous membrane, which may further show marked evidence of atrophy of the glands, with or without excessive thickening of the submucosa. The liver is nearly always slightly enlarged, and fattily degenerated. In definite cases the outer zone of the lobules is pigmented by an accumulation of free iron (hæmosiderin) within the cells and around the capillaries. The presence of this substance is revealed by treating sections with sulphide of ammonium (not a very trustworthy test) or ferrocyanide of potassium, and its discovery by Quincke led to the hypothesis of the disease that is now mostly adopted. The gall-bladder contains dark bile. The spleen may be slightly enlarged, but often it is quite small; in colour and consistence it varies, being often pale, sometimes soft, or again rather indurated. In this organ, too, but never to so marked an extent as in the liver, granules of ferruginous pigment have been met with. The pancreas and suprarenals show no changes. The intestinal plexuses of nerves and the great abdominal ganglia have been found to exhibit evidence of degeneration. The mesenteric and other lymphatic glands are not as a rule affected. The kidneys are smooth and pale, but pigment granules have been found in the cells of the cortical tubules. As regards the nervous system, there may be subarachnoid hæmorrhage on the surface of the brain. The brain itself is strikingly exsanguine. It is instructive to note that although sinus thrombosis has been observed in chlorosis, it has not been recorded in pernicious anæmia. Cerebral hæmorrhage, however, may be present. In the spinal cord, even in cases which have not presented any symptoms of such disease during life, tracts of sclerosis have been met with in the white matter; they are irregularly distributed, sometimes involving the whole of the posterior columns, together with the lateral and anterior, but generally sparing the nerve-roots and the gray matter. Another change is that of miliary sclerosis or minute hæmorrhagic foci scattered irregularly throughout the substance of the cord. Notable changes have been found in the marrow of the long bones, consisting in a reversion to the fœtal type of red marrow; when first met with, this conversion was thought to be distinctive, and no doubt it indicates increase of the hæmogenetic function; but it is also present in anæmia due to hæmorrhage, and may be absent in the disease under consideration.

Pathology.—The interpretation of the clinical and pathological facts of so obscure a disease as pernicious anæmia could not fail to arouse widespread interest, and the attempt to afford a rational explanation of its origin has led to much speculation and to more or less thorough investigation. There is no need to dwell further upon a matter already touched upon in speaking of etiology, but it may be said that for coherence and reasonableness of doctrine there has been no more satisfactory exposition than that given by Dr. William Hunter in his numerous essays upon this disease in particular and upon the subject of blood-destruction in general. For, although in some respects it may be necessary to admit certain modifications in his argument, seeing that some of its premisses are not yet verified, yet it cannot be doubted that his contention for the specificity of pernicious anæmia, as a disorder consisting in hæmolysis, affords so far a satisfactory explanation of the phenomena. It has further enabled us to eliminate from the category of pernicious anæmia many anæmias which are strictly secondary; closely as they may simulate the primary disease in clinical features, blood changes, and visceral lesions. Nor is it warrantable to include within the class such cases as those of fatally progressive anæmia, associated with the presence of intestinal parasites; unless it can be shown that they depend on the same kind of hæmolysis that underlies the primary malady.

Pernicious anæmia, then, signifies a definite group of clinical and pathological phenomena dependent upon a special form of blood-destruction, or hæmolysis, induced by toxic agents absorbed from the gastro-intestinal tract. The grounds for this conclusion may be briefly summed up as follows:—(i.) There is abundant proof that hæmolysis does take place in this disease. This is shown by the condition of the blood, its deformed and disintegrating corpuscles, the readiness with which the hæmoglobin escapes from them, and the abundance of microcytes. (ii.) The presence of an excess of pigment in the liver, spleen, and kidneys; this pigment being in the form of iron granules very loosely combined in the cells; whilst the elimination of iron and occasional excess of pathological pigments in the urine further support the hæmolytic view. (iii.) This hæmolysis takes place within the area of the portal circulation and not in that of the systemic; as indicated by the condition of the spleen, the accumulation of the hæmoglobin detritus (hæmosiderin) within the hepatic cells of the outer zone of the lobules, and by the absence of hæmoglobinuria. For Hunter's researches prove that when hæmolysis takes place in the general circulation hæmoglobinuria occurs; but in pernicious anæmia the pigment, if eliminated by the kidney, appears in the form of granules of iron pigment, or as an excess of other pigmentary matter variously regarded as "pathological urobilin" (Hunter, M'Munn) or as a mixture of "urobilin" and "hæmatoporphyrin" (Gowlland Hopkins). Moreover, iron granules have been found in the tubules of the kidney in renal casts and epithelia. (iv.) Such a disintegration of the blood can take place in the portal system (possibly mainly in the spleen), as is shown by Hunter's experiments with such hæmolytic poisons as toluylendiamin. The source of this hypothetical toxin is reasonably considered to be the gastro-intestinal tract, and that it is of bacterial origin is almost equally probable.

The conception that has thus been framed of the nature of pernicious anæmia is rendered more convincing by the fact that in the forms of anæmia which most nearly approximate to it—such as those due to other toxic agencies, to prolonged and repeated hæmorrhages, to blood parasites as in malaria, to cancer, or to syphilis—so marked an excess of iron in the viscera is never found, especially in the liver, as is found in this disease. Thus Hunter gives 0·7 as the average percentage of iron found by various investigators in the liver in pernicious anæmia, as against 0·078 or 0·12 for other diseases; whilst the late Dr. Beavan Rake found, from an examination of five cases of anchylostomiasis, that in this affection (which so closely simulates pernicious anæmia as to have led to the opinion that, like pernicious anæmia, it may perhaps depend upon hæmolysis in the manner peculiar to this disease) it was only 0·1 per cent. Dr. Rake, it is to be noted, adhered to the opinion that the anæmia of anchylostomiasis solely depends upon the hæmorrhages produced by the parasite from the intestinal wall.

At the same time, in the present state of knowledge it is impossible to avoid the conclusion that a hæmolytic process closely akin to that of pernicious anæmia may occasionally intervene in the course of grave organic disease, and especially of chronic gastro-intestinal disease. If this be so, however, it would be no longer correct to speak of such an exceptional and, so to speak, accidental supervention of the pernicious process as a "symptomatic" anæmia. The secondary affection should rather be regarded as a complication than as a regular feature of the original disease.

The view of the nature of anæmia alternative to that of increased hæmolysis—one, too, which in point of time preceded the promulgation of the latter—is that of disordered hæmogenesis, as suggested by the remarkable reversion of the bone-marrow to its fœtal condition originally observed by Pepper and Cohnheim, and since verified by many other observers. The significance of these changes has been materially affected by the recognition of the part played by hæmolysis in the disease, as well as by the fact that they are not invariably met with, nor differ in kind, if they do in degree, from the changes in the marrow which ensue on anæmia experimentally produced by bleeding. It may be that a place should be retained in nosology for a "myelogenic anæmia," but if so it must stand apart from pernicious anæmia as now understood. Whenever these marrow changes are met with side by side with the characteristic hæmolytic features of pernicious anæmia they are more likely to be of a secondary nature, indicating an effort on the part of the hæmogenetic organ to repair the waste that is in progress elsewhere. And that the marrow should exhibit these changes in some cases and not in others may not be more remarkable than that the spleen should be swollen and apparently in an active state of hæmolysis in some cases, shrunken and inactive in others. The course of the malady suggests an inconstant and variable hæmolytic activity, and it may well be that this is paralleled by varying degrees of hæmogenetic action. In one of the most recent and careful studies of these marrow changes in pernicious anæmia the conditions obtaining in the several stages of the disease are described; the earlier changes are similar to those met with after hæmorrhage, the later are characterised by great abundance of large nucleated corpuscles peculiar to pernicious anæmia, and suggestive of a reversion to the embryonic type. But both must be considered secondary to the anæmic state (Muir). Dr. Muir is careful to add, however, that "whether or not there are any cases of fatal anæmia, in which marrow lesion is a primary factor, I am not prepared to say. The question ought to be kept an open one, and in all such cases the condition of the marrow should be carefully inquired into, along with any changes in other organs which point to a process of blood-destruction."

There remains another aspect, ably described by Prof. Stockman, in which pernicious anæmia has been regarded which, if correct, would dethrone the disease from the position which it has attained, and relegate it to that of a sequel or result of any form of anæmia of whatever origin. It is based on the fact that the anæmic state, if long continued, provokes fatty degeneration of the walls of the blood-vessels, and thus promotes a liability to multiple hæmorrhages, which in their turn intensify the anæmia and the proneness to bleed; and further, that the effects of larger hæmorrhage are pathologically indistinguishable from those which in anæmia take place within the tissues and organs. It cannot be denied that such an explanation of the nature of pernicious anæmia, if substantiated, would get rid of many of our present difficulties, for it bridges over the gap between the protopathic and deuteropathic forms of progressive anæmia by referring them all to the same immediate cause. Yet as Addison could find no adequate cause for the production of "idiopathic" anæmia, so too is it difficult to believe that the capillary hæmorrhages which characterise pernicious anæmia are in the majority of cases sufficient to induce the extreme degree of oligocythæmia, and the indubitable evidences of hæmolysis which the disease exhibits. At the same time, Stockman's thesis is one which deserves most careful study, for he does not hesitate to deal with the whole of the pathological and chemical evidence put forward by the advocates of the hæmolytic doctrine.

This doctrine also assumes the operation of a specific toxic agency; and it is interesting to note that those who have studied the degenerative changes in the spinal cord, which are apparently more common in pernicious anæmia than might be supposed from the clinical phenomena, believe also that these changes are best explained by a toxic influence, although some of them are manifestly the result of capillary hæmorrhages. In accordance with the prevalent views upon the subject, the nature of pernicious anæmia is expressed in the classification of anæmic disorders put forward by Crozier Griffith and Musser. The anæmias are divided by them into two main groups—the cytogenic and the non-cytogenic. Of the latter there are two classes—the hæmolytic and the oligocythæmic. The *hæmolytic* comprise—(i.) Pernicious anæmia; (ii.) other toxic anæmias; (iii.) chlorosis; (iv.) parasitic anæmias (some forms). The *oligocythæmic* include—(i.) Parasitic anæmias (some forms); (ii.) post-hæmorrhagic anæmia; (iii.) anæmia from loss of albumin; (iv.) anæmia from malnutrition. Such a division is only provisional, but it recognises at least that pernicious anæmia is entitled to a distinctive place in nosology.

Diagnosis.—The diagnosis of pernicious anæmia does not rest upon any very certain basis, for although, generally speaking, this diagnosis may be justified in the presence of a case of progressive anæmia, arising insidiously, without adequate discoverable cause, and exhibiting the characteristic changes in the blood already described, it must yet be borne in mind, first, that sometimes an adequate cause does exist, but cannot be discovered, and, secondly, that the blood changes are not in themselves pathognomonic. At the same time, when it is considered that the clinical phenomena may be simulated by profound anæmia of secondary origin, no endeavour should be lacking to ascertain whether there is or is not some underlying disease. The task is rendered easier as the number of conditions which are known to give rise to so grave an anæmia are not large. The most likely are malignant disease, especially of the stomach, and advanced syphilis. In such cases attention to the course of the symptoms and the history of the patient may assist in effecting a separation. The anæmia which is sometimes so marked a feature of chronic parenchymatous nephritis is seldom likely to be mistaken for pernicious anæmia, in view of the predominance of definite signs of the renal affection. Malignant endocarditis is more likely to be mistaken for pernicious anæmia, for here anæmia may be pronounced, whilst the cardiac murmur, and even the pyrexia and the cutaneous or other hæmorrhages, may be looked upon as indications of pernicious anæmia. But an examination of the blood will determine the true character of the affection. Purpura and hæmophilia are hardly likely to be mistaken for pernicious anæmia.

From chlorosis, which shares with pernicious anæmia the title of a protopathic anæmia, the differences are fairly well marked. Between the two affections the clinical diagnosis, apart from considerations of age and sex, is to be made by examination of the blood. In chlorosis the reduction in hæmoglobin is always vastly greater proportionately than the reduction in the number of corpuscles; the chlorotic blood does not show such marked evidence of poikilocytosis, or so many microcytes as that of pernicious anæmia. More difficulty may be experienced in discriminating the anæmias due to intestinal parasites, such as the anchylostomum or the bothriocephalus, for there are no distinctive features either in the blood or in the symptoms which would serve to distinguish them. It is possible that the urine might afford means for the diagnosis, but it is by no means certain that all cases of pernicious anæmia exhibit that excretion of "pathological urobilin" which is held to characterise the affection. In fact, nothing but a thorough examination of the fæces for the detection of the ova of these parasites can suffice to exclude them, and the fortunate issue of anthelmintic treatment may clinch a diagnosis so made. From splenic anæmia the diagnosis is to be made by the marked and progressive enlargement of the spleen in this disease, as well as by the comparatively small degree of oligocythæmia. In leukæmia the blood condition is manifestly the diagnostic criterion.

Prognosis.—The outlook in a case of established pernicious anæmia is very grave. It would, however, be too much to say that it must necessarily end fatally, for cases are recorded where the patients were restored to health and remained in health for years afterwards. It is nevertheless only too true that what has often been regarded as recovery has proved to be but a temporary rally, however remarkable in degree; the oligocythæmia almost disappearing and the distressing symptoms entirely passing away. Such apparent recoveries often, after an interval of months, give way to relapse, and the end may come in the first or some subsequent recurrence of the illness. This character is so common a feature, after more than one method of treatment, as to raise doubts whether the rally is wholly attributable to the latter, whether, that is, there may not be a "relapsing" form of the disease.

Treatment.—Pernicious anæmia then, as its name implies, tends ordinarily to run a downward course, often uninfluenced by any treatment that may be adopted; and frequently, too, when marked improvement has followed the use of certain remedies, a relapse has occurred in which the same means are no longer successful. As a recent writer (Dieballa) points out, it may be that much depends on the amount of recuperative power still residing in the blood-forming organs. If the blood-destruction can be arrested, or if the hæmogenetic function can be stimulated, then there is hope that the blood will be restored to the normal by the natural power of regeneration. So far as can be judged, most of the therapeutic efforts that have been attended by success, temporary as this may have been, have had for their aim the second of these two conditions; but treatment based upon the theory of intestinal intoxication has not been wanting.

The general principles of the treatment of anæmia naturally apply in this disease with especial force. Rest, bodily and mental, and the avoidance of excitement are not only essential, but are often spontaneously sought by the patient, who is disinclined for exertion of any sort. Removal to pure air and healthy surroundings, when the conditions of the patient's ordinary life are without these benefits, are too obvious to require mention; whilst for the well-to-do much benefit may accrue, if the disease be not advanced, from a winter sojourn in a more equable and brighter climate than this country affords. The matter of diet is one of the greatest importance. The anorexia, nausea and tendency to diarrhœa, which often mark the early stages, make this a matter of difficulty. Experience proves that nitrogenous foods are ill borne, and the dietary therefore must in the main be limited to milk, vegetable and farinaceous foods. It may be necessary to have recourse to peptonised and other easily digestible preparations. When tolerated, pounded raw meat or meat juice and bone-marrow may be taken in small quantities, on bread or toast, with advantage. But the disinclination for food of any kind may be so great as to render it very difficult to supply adequate nourishment. Small quantities of alcohol in the form of claret or burgundy may prove of value as aids to digestion.

In a disease which is primarily dependent on destruction of the elements of the blood, hæmatinic remedies may reasonably be adopted. It is remarkable, however, that the chief of these—iron—is in the great majority of cases quite inoperative. In this respect the difference between chlorosis and pernicious anæmia is so striking as to suggest at once that the pathogeny of these two forms of primary anæmia is totally different; evidence of the inefficacy of iron in the latter is probably to be found in the fact that the system still retains its iron; in greater amount, indeed, than can be properly utilised. At the same time, there are a few cases on record in which iron seemed to do good; the free and frequent administration of the perchloride has been especially advocated. It is possible, however, that the value of this preparation may not depend on the ferruginous element (*vide* p. 515).

Very different is the experience of the value of arsenic, the introduction of which in the treatment of pernicious anæmia we owe to Dr. Byrom Bramwell. Administered in gradually increasing dose, it is generally well tolerated by these subjects, and although by no means invariably successful in causing improvement, yet this does often follow, and can hardly be ascribed to anything else than the specific action of the drug upon hæmatogenesis. On the other hand, if there be a natural tendency in the disease towards a temporary rally, it is difficult to estimate the precise share in this which is to be ascribed to the action of the remedy. At the same time, the improvement, when it does take place, follows too closely upon the adoption of the treatment to afford room for scepticism. Although of all remedies arsenic has proved to be most often followed by manifest improvement, yet even in cases where the benefit has been striking, relapses have occurred sooner or later; and it often happens that on the second occasion the drug seems to have lost its efficacy, and other measures have to be devised. The arsenic may be given in the form of Fowler's solution, or of the liquor arsenici hydrochloricus, beginning with doses of 2 to 3 minims, which are gradually increased to 18 minims, according to the tolerance of the subject. The arseniate of iron pill is a convenient form. It is well to continue the drug for some time after the signs of improvement are manifest.

Cases of recovery have also been recorded after the use of antiseptic drugs, such as salol, β-naphthol, salicylate of bismuth, and the like; their efficacy being ascribed to their direct antagonism to the supposed fermentative processes which yield a hæmolytic poison in the intestine. A notable instance of rapid recovery, which, moreover, was sustained for a long time, has been recorded by Dr. Gibson; it followed the prescription of β-naphthol, after the failure of arsenic. In another case, where for nearly four months a variety of remedies had been vainly tried in turn—namely, ferratin, bone-marrow, oxygen, arsenic, iron, and quinine—the administration of salol (continued with occasional intermissions for three months) produced a restoration in the corpuscular richness which seemed to have been due to the change of remedy (Dieballa).

Other isolated cases of similar good results from this line of practice, including that of lavage of stomach and intestinal irrigation, have been recorded, although not seldom the same methods have led to no good result.

Indeed there are few maladies in which the results of treatment are more capricious. It is impossible to prophesy in any given case whether any given remedy will be useful or not. Dieballa, in commenting on the case of recovery after the administration of salol, points out that the comparatively slight diminution of leucocytes and the persistence of a normal proportion of eosinophilous cells in the blood, as shown in that case, may afford a clue to the integrity of the blood-forming organs, and may justify expectation of adequate recovery if the hæmolysis can be arrested.

We have yet to discover the reasons for the frequent failure of a remedy in one case and for its success in another. Thus the success attending the use of red marrow, introduced by Prof. Fraser, has been repeated by some physicians, but never attained by others. A like diversity of experience is to be found in the records of cases treated, often, no doubt, in the last resort, by blood transfusion or saline injections. Excellent and even remarkable results of transfusion have been published—amongst others by Quincke, Brakenridge, and Affleck—results which often seem quite out of proportion to the amount of blood injected; as if the healthy serum had exerted some specific effect, either in stimulating hæmogenesis or, as some think, possibly by exerting an antitoxic influence upon the (assumed) hæmolytic virus. Yet in very many cases this measure has proved futile.

Thus in pernicious anæmia, where the therapeutical results are so varied and conflicting, it is impossible to frame any uniform plan of procedure; and the inconstancy of therapeutical results may be taken as evidence that there is much yet to learn of the intimate pathology of the disease. All that can be done in the presence of the progressive blood-destruction is to make trial of each of the several remedial measures that have been found at times to be efficacious; of these I would place first the administration of arsenic, and next to it that of intestinal antiseptics.

Sidney Coupland.

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The above select list is by no means exhaustive of the very copious literature of the subject, which of recent years has been greatly added to. Nor does it take account of the numerous articles and monographs upon the various forms of parasitic anæmia, notably anchylostomiasis and bothriocephalus anæmia.

S. C.

SPLENIC ANÆMIA

There is a form of profound anæmia, progressive in character, ending fatally, generally of no long duration, associated with great enlargement of the spleen, but without leucocytosis or enlarged glands. Splenic anæmia is the name by which the disease is best known in this country ; but it has also been called *splenic cachexia*, *splenic pseudo-leucæmia*, *lymphadenoma splenicum*, and *spleno-megalie primitive ;* under the last name chiefly it is described in French literature.

To Banti, who wrote in 1882, is due the credit of drawing special attention to this malady. In 1891 Bruhl published an exhaustive article on the subject, bringing together all the cases that he was able to find recorded up to that date. The other contributions consist for the most part of accounts of isolated cases.

The total number of cases recorded is still small, and probably does not exceed thirty, including the fourteen cases upon which Bruhl's paper was founded.

Symptoms and signs.—The disease may be divided into three stages: in the initial stage the symptoms are those of extreme anæmia, with great loss of muscular power and some wasting of muscle ; though usually without emaciation. As in this stage the disease presents no specific features it can rarely be recognised. The second stage is characterised by progressive enlargement of the spleen, and by attacks of severe pain in the splenic region ; the anæmia is more profound, the loss of strength is extreme, and the patients are liable to repeated attacks of bleeding, especially from the nose; the temperature is now usually raised and of hectic character, reaching 102° or more in the evening. It is in this second stage that the disease is first recognised.

In the last stage the condition is one of progressive asthenia which ends in death; there is in it nothing especially characteristic.

Throughout the disease most of the symptoms present but few peculiarities, for they do not differ from those which occur in any form of profound anæmia. Thus there is general pallor and loss of strength, and great weakness and dilatation of the heart with its consequences; namely, shortness of breath, palpitation and pain, all made worse by exertion, together with the usual hæmic murmurs. The pulse and respiration are readily accelerated, especially on effort or excitement; and there may be some œdema of the feet.

The hæmorrhagic condition in the latter stages is only remarkable in that it is more than usually pronounced.

This form of anæmia is sometimes said to be of the chlorotic type; as there is little or no emaciation, and the reduction in the hæmoglobin is greater than the reduction in the number of red blood cells would account for.

With a disease so rare as splenic anæmia the best description of the disease will be an account of a case:—

A man aged thirty-six presented himself with extreme anæmia, raised temperature, and a large spleen. The case looked like one of splenic leucocythæmia, but examination of the blood showed no increase of white cells.

The patient had been well till twelve months before he came under observation, when a tooth had been extracted; this operation was followed by profuse bleeding, which lasted several days: from that time onwards he became gradually weaker and thinner, and suffered from repeated epistaxis.

The patient was extremely pale, cachectic, and somewhat sallow; his cheeks were flushed, and the temperature, on the evening of admission, reached 103°. He was constantly spitting up a little blood, which came from the back of the pharynx or from the nose. The respiratory organs were normal. The heart was somewhat dilated, with a blowing, systolic murmur audible over the whole precordium and loud at the pulmonary area; there was increased pulsation in the vessels of the neck; the pulse was 96, of low pressure, but fair volume.

The liver was somewhat enlarged, extending from the upper border of the fifth rib to an inch and a half below the costal arch.

The spleen was greatly enlarged, and extended from a point four inches above the costal arch downwards to an inch above the anterior spine of the ilium on the left side. It was smooth on the surface, moved freely in respiration, but was tender to touch.

The urine was normal.

The left pupil was a little larger than the right. Ophthalmoscopic examination showed the retinæ to be normal.

There was no tenderness of the bones and no enlargement of lymphatic glands.

On examination of the blood the red corpuscles were found to number

2,055,000, and the white corpuscles 50,000, chiefly lymphocytes; the hæmoglobin was only 25 per cent of the normal. There was no poikilocytosis or other changes in the cells, red or white.

Three weeks later another examination of the blood was made. The red cells had fallen to 1,900,000, while the white corpuscles numbered 58,000; the anæmia had progressed and the strength failed, there had been occasional attacks of abdominal pain, sometimes in the splenic region, sometimes more diffuse, but not very severe; the temperature had remained of a hectic character, rising to about 103° every evening, as shown upon the chart, and the patient had one or two attacks of epistaxis. The blood was examined for micro-organisms, and none found.

A week later the eyes were examined again, and a large hæmorrhage was found in the left retina.

A few days later the patient became very hoarse, dyspnœa increased rapidly, œdema of the larynx was diagnosed, and tracheotomy performed with great relief. The temperature remained high, and after the operation reached 105°. There was a good deal of oozing from the incision after the operation, and five days later a sudden hæmorrhage took place from the wound; blood was sucked into the trachea, and the patient was suffocated.

The necropsy showed a large spleen weighing 76 oz.; it contained one small infarct. On microscopical examination the Malpighian bodies were seen to be much diminished in size and badly formed, and there was a slight general increase in the trabecular tissue. The liver also was enlarged, weighed 93 oz., and was slightly cirrhotic. The heart was dilated and weighed 12 oz.; all its cavities contained post-mortem clots; there was a small vegetation, as large as a pea, on one of the aortic valves. The muscular substance was not fatty. The larynx was still somewhat œdematous.

A full account of this case is given in the *Transactions of the Medical and Chirurgical Society*, vol. lxxix.

Williamson's case is also an interesting one to compare with that just recorded:—

It occurred in a lad aged nine who, for two years, had been growing increasingly pale and anæmic, and had suffered for the last twelve months from fortnightly attacks of epistaxis. He was extremely anæmic and had a very large spleen. The examination of the blood was as follows: red cells, 3,540,000; white, 4000; hæmoglobin, 22 per cent.

Four months later the red cells were reduced to 2,510,000, white 2000. A month later the following report of the blood was made. There were a number of poikilocytes, no eosinophile cells, no large mononuclear or granular cells, but a slight increase of lymphocytes. The temperature for the last six months of life was very irregular, with marked daily oscillations reaching to 101° and 102° at times. The patient died with an attack of acute peritonitis.

The necropsy showed a very large spleen weighing 40 oz.; the liver reached two inches below the ribs and weighed 44 oz. There was a small serous effusion in the pericardium, and some recent vegetations on the mitral valve. A small ulcer was found in the small intestines, which had perforated and caused purulent peritonitis.

In the spleen the fibrous trabeculæ were increased in thickness, there was an enormous number of large nucleated cells each containing several red blood-cells. The Malpighian bodies had undergone fibroid change and the lymphoid cells were few. There was a slight iron reaction in the fibrous trabeculæ and in the Malpighian bodies, but none in the spleen pulp. Micro-organisms were looked for and none found.

The liver showed a little increase of connective tissue, and gave a very slight iron reaction.

The bone-marrow was dark purple-red in colour, and showed a marked absence of fat. It contained large cells enclosing several red blood-cells, as in the spleen.

With these general remarks we may now pass on to the review of the more special features of the disease.

The affection occurs with much greater frequency in men than in women; thus, out of 24 cases 19 occurred in men and 5 only in women, that is, 4 males to 1 female.

In respect of age the affection seems to be fairly equally distributed through all the age-periods of adult life; thus, of 22 cases 13 occurred between the ages of 20 and 50, and these were fairly equally distributed within this period. Cases, however, occur in children and also in old persons; the youngest on the list was aged 9, and the oldest 72 years.

In infancy and very young childhood I do not know that there is any undoubted case on record; and, although anæmia and large spleens are by no means uncommon in these early years of life—cases which have been described by some writers under the name of splenic anæmia—still a review of these cases and the course they run shows, I think, that we have to deal in them with disease of an entirely different kind.

The Blood. — The blood shows no pathognomonic changes. The condition is simply that of profound anæmia. The red cells are diminished to one-fourth their normal number or less, and their form is preserved, though they are a little reduced in size; usually there is no poikilocytosis. The cells are poor in hæmoglobin, which is reduced to one-quarter or one-sixth, and the loss is far in excess of the diminution of red blood cells. Occasionally there is a slight increase in the white cell, but not more than the fever or some intercurrent malady would account for. As the disease advances there is a continuous reduction in the number of red cells, as is shown in the following scheme (taken from Dr. F. Taylor's paper):—

	Red Cells.	White.	Hæmoglobin.
Sept. 27	3,000,000	no increase	35 per cent
Oct. 4	2,600,000	1 to 300	35 ,,
,, 17	2,400,000	...	30-35 ,,
,, 25	2,200,000	increased	...
,, 27	1,550,000	1 to 70	...
Nov. 11	1,370,000	1 to 28	28 ,,

The blood has been carefully examined in several instances for micro-organisms, microscopically as well as by cultivation, but without success.

In many of the cases, especially those which were recorded some years ago, the blood examination was not as systematic as it would be in the present day. In my own case it was made under the supervision of Professor Kanthack, and good accounts of the blood are also given in the cases described by Williamson and Taylor.

Williamson's case is peculiar in the fact that there was poikilocytosis, which, as already stated, is usually absent.

With reference to the increased number of leucocytes which is sometimes observed, especially where the temperature is high, it must be remarked that the increase is chiefly due to lymphocytes; that there is none of the changes in the eosinophile and other cells which are characteristic of leucocythæmia, and, finally, that the increase, being due in great measure to the extraordinary diminution in the number of red blood cells, is rather relative than absolute.

The Spleen.—The spleen is considerably enlarged and tender, and the surface usually feels smooth; but sometimes it may be uneven. Signs of local peritonitis in the splenic region may be present, or of left basic pleurisy, in both cases due to inflammation spreading from the spleen. Some general peritoneal effusion has been also met with.

The spleen may extend beyond the umbilicus and as far as the crest of the ilium, and be of considerable weight, as will be further described under the morbid anatomy. The spleen is observed, as the case progresses, to increase in size, especially during the exacerbations; but occasionally between the attacks it seems to become for the time smaller. The recurrent attacks of pain in the splenic region, apparently due to peri-splenitis, are often the cause of very great suffering.

The Liver.—The liver is often somewhat enlarged also, and may extend, as in the case described, from the fifth rib in the nipple line to an inch or two below the costal arch. It is sometimes associated with slight jaundice, and there may be some pain felt in this region from time to time.

The Digestive system.—The digestion is considerably disturbed and the appetite lost. There is a good deal of nausea and sometimes obstinate vomiting; this may occur in such paroxysmal attacks as almost to constitute crises, and they often coincide with attacks of abdominal pain. Constipation is usually troublesome; but occasionally diarrhœa is present, and this may be almost dysenteric in character, with tenesmus and discharge of bloody mucus: in one or two instances there has been free hæmorrhage from the bowels.

Hæmorrhages.—The tendency to bleeding is pronounced: the hæmorrhages are usually of slight degree and of the nature of oozing; but they frequently recur, are very difficult to control, and add greatly to the anæmia. Profuse hæmorrhages from any part are uncommon, but they are recorded as occurring both from the stomach and from the bowel, or, as in my case, from a wound; in each instance they proved fatal.

Epistaxis is very frequent and usually one of the earliest symptoms. Though rarely profuse, it is of importance on account of its frequent occurrence: it may, however, be so severe as to require plugging of the nares. Oozing from the gums, again, is by no means uncommon, and it is most difficult to check.

Hæmoptysis and hæmaturia have been met with also, but they are rarer than other forms of hæmorrhage.

From the gastro-intestinal organs hæmorrhage is rare, and, if in any large amount, it is probably associated with some secondary lesion. A case, however, is recorded by Dr. Douglas Stanley, in which, although profuse and fatal hæmatemesis took place, no lesion in the stomach was found after death; in another case, however, a gastric ulcer was present. Müller records a case of fatal hæmorrhage from the bowels, and there an ulcer was found in the small intestines.

In the skin small petechiæ on the lower limbs are common, especially in patients who are not in bed; but they are of no special significance. A purpuric eruption of greater degree than this is not described.

Into or behind the retina hæmorrhages may occur, no doubt, as they do in other forms of anæmia; but I do not know any instance of it except that which I have described.

The Temperature.—Bruhl states that fever is unusual, but in many of the recorded cases the temperature reached a considerable height. It has been of the nature of an irregular hectic, rising even to 103° or 104° every evening (cf. Chart).

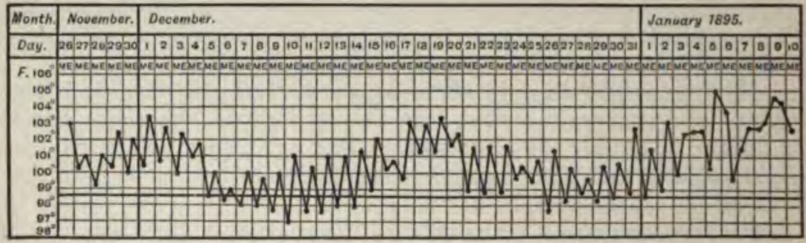

CHART 5.—Maximum (evening) and minimum (morning) temperature daily.

It is probable that something depends upon the stage which the disease has reached; that in the early stages the temperature may not be raised, or be even subnormal, while in the later stages, when the disease is actively progressing, it may be high.

At any rate the rise of temperature, when it occurs, appears to be part of the disease, and not to be connected with any complication.

The *nervous system* yields no special symptoms.

Urinary changes are indefinite and vary much, but sometimes albumin is present in small amount.

The *circulatory system* presents only such changes as are common to all forms of anæmia.

In the *skin* pigmentary changes have been described, but as in the majority of these cases arsenic had been administered in large doses and for some time, they were very probably due to this drug.

Morbid anatomy.—The general pallor of the skin and of all organs, the flabbiness of the muscles, the dilatation and degeneration of the heart are common to all forms of anæmia.

The *panniculus adiposus* is usually well preserved; and in this respect—namely, in the absence of marked emaciation—the body contrasts with that of malignant disease of the spleen, a still rarer condition a case of which, however, has been recorded under the name of splenic anæmia.

The Spleen.—The most obvious morbid change is the enlargement of the spleen. This organ may be 12 inches long or somewhat more, and may weigh from 2 lbs. to 7 lbs. It is firm, reddish brown in colour, with well-marked notches and, occasionally, irregularities on the surface. It is surrounded by a thickened capsule, which may be adherent either to the diaphragm or to the parts about. On section it looks as if there were a general hypertrophy of the organ; but occasionally there are one or two hæmorrhagic infarcts in it. The surface of the section is dry and yields little juice; it shows grayish streaks or fine granulations; the former, on microscopical examination, prove to be thickened trabeculæ, the latter fibrotic Malpighian corpuscles. The Malpighian bodies are stated, in many of the cases, to have been the seat of marked changes, the central artery being thickened, the corpuscle shrunken and shrivelled, and sometimes surrounded by a capsule of fibrous tissue. With the thickening of the trabeculæ there has been great disappearance of the spleen cells and dilatation of the veins. In Williamson's case many nucleated cells, each containing six to ten red blood-cells, were found in the splenic pulp; but in the majority of cases no special changes are present in it.

The lesions in the spleen then appear to be: (i.) fibrosis of the organ; (ii.) disappearance of the pulp; and (iii.) the cirrhosis and atrophy of the Malpighian bodies. This last is regarded as the characteristic and most important lesion, and is the exact opposite of the state found in leucocythæmia, in which disease the Malpighian bodies are, as a rule, hypertrophied.

The *liver* is slightly cirrhosed and is much pigmented. The cells are misshapen, atrophied, and often granular. The iron reaction is usually absent; it is only described as present in Williamson's case, and then it was insignificant in degree.

The *pancreas* also, in some cases, has been found indurated; but this lesion is not constant, and probably, therefore, stands in no direct relation to the disease.

The *lymphatic glands* are normal.

The *bone-marrow* also is usually normal; but in one or two instances it has been described as red and infiltrated with leucocytes.

The *heart* is dilated, the muscular substance flabby and occasionally fatty. In one or two cases minute vegetations have been found upon the valves; as in my own case, in Williamson's, and in Strümpel's. In connection with these vegetations may be mentioned the infarcts which have been found now and then in different organs, notably in the spleen and in the kidneys.

These vegetations and infarcts deserve attention because undue significance has been attached to them. They are indeed absent in all the recorded cases except those which I have mentioned; and in these they were obviously accidental and formed no essential part of the disease.

When hæmorrhage has occurred during life the traces of it will be seen after death; but, besides this, hæmorrhages of small size, petechiæ for the most part, may be found very widely distributed in many parts of the body—in the lungs, pleura, pericardium, and even in the brain; but these, again, are of no special significance, for they are found in all cases of profound anæmia in which the hæmorrhagic tendency has been well marked. For the most part, no doubt, they arise shortly before death.

Pathogeny.—That these cases of anæmia form a definite clinical group must, I think, be regarded as fully established. The cause or pathogeny of the disease is hitherto a matter of hypothesis and opinion.

In the second stage, when the spleen is enlarged, and especially when fever is present, the cases bear a close clinical resemblance to many septic diseases, and especially, perhaps, to some forms of malignant ague. This would suggest that some parasite or septic organism is present in the blood. Careful examinations, however, both of the blood and of the spleen, have been made and have proved negative. In my case the results were negative, as they were also in two cases investigated by Sciola and Carta. In one of them, that of a girl aged 13, injections were made in the spleen of different animals with blood from the spleen of the patient, and after removal of the spleen with splenic substance; in all cases without result. In another case, that of a woman aged 29, cultivation experiments were made with the extirpated spleen on different media without result.

So far, then, as our present knowledge goes, bacteriological investigations have yielded no new facts.

The enlargement of the spleen observed during life, standing in close relation as it does to the severity of the disease and the peculiar morbid appearances discovered after death, have suggested that the disease is directly due to the affection of the spleen itself; that is to say, to the peculiar atrophy of the Malpighian corpuscles. So far, however, the number of cases is too small to determine whether the peculiar changes described in the spleen are constant, and we must wait until this is established before we found a hypothesis upon such a pathological basis.

Complications.—The complications may be divided into two groups: first, those which are obviously accidental, and, secondly, those which stand in some possible relation to the disease itself.

In the first group we have instances of death produced by pneumonia and by bronchitis.

In the second group may be placed peritonitis or abscess in the region of the spleen, left-sided pleurisy perhaps, and the severe hæmorrhage from the stomach or intestines which has been recorded in a few cases.

The course of the disease is usually continuously progressive: there may be periods of temporary arrest, or possibly even of improvement; but in the end relapse occurs, and the result is the same.

The duration of the disease.—The disease is not of long duration, from six months to two years, rarely longer; yet Müller records a case which lasted four and a half years. It is possible that there may be cases of even shorter duration than six months, for Ebstein records one, under the name of Pseudoleukæmia splenica, but the nature of these rapid cases is somewhat doubtful.

Prognosis.—Prognosis in respect of recovery is hopeless; in respect of duration of life is bad; and in respect of the immediate risks to life must be determined in each case by the condition of the patient.

The mode of death, as a rule, is by progressive asthenia; though the end may come suddenly with cardiac syncope, or it may be determined by hæmorrhage, as in the cases referred to.

Diagnosis.—In the early stage diagnosis is impossible; in the later stages it is comparatively easy on account of the enlargement of the spleen.

1. From *pernicious anæmia* splenic anæmia is distinguished by the enlargement of the spleen, as well as by the condition of the blood; though in a clinical sense the anæmia is pernicious, being progressive and fatal.

It is especially from other forms of profound anæmia in which the spleen is enlarged that the diagnosis has to be made. Foremost among these stand leucocythæmia and Hodgkin's disease.

2. In *leucocythæmia* the diagnosis is determined by the characteristic blood changes which are absent in splenic anæmia.

3. In *Hodgkin's disease* the anæmia is usually not so profound, there is a greater enlargement of the liver, and the lymphatic glands are affected.

4. In *malignant disease of the spleen* emaciation is a prominent symptom, and the wasting advances as the disease progresses; there may be secondary growths elsewhere, and there is no rise of temperature.

5. In *pernicious ague* or *malarial fever* the temperature is of a more intermittent and irregular character; moreover, the presence of the parasites in the blood and the history of the case will fix the diagnosis.

6. *Syphilitic disease of the spleen* may possibly cause some difficulty as in Coupland's case; but as a rule the history and other evidences of syphilis will help the diagnosis.

7. With *tuberculous disease of the spleen* there is generally marked wasting and the evidence of tuberculous mischief in other organs; while as a rule the anæmia is not so profound.

8. In *cirrhosis of the liver*, with secondary enlargement of the spleen,

there is again not so much anæmia, and the ordinary signs and history of cirrhosis of the liver are obtained.

9. In *children* anæmia and enlargement of the spleen are not uncommonly associated. The causes are many, but among them it appears that splenic anæmia, in the sense in which it is here used, is not to be reckoned; for a conclusive instance of the disease under the age of nine years has not yet been recorded. The course of these cases in children is also different, for most of them end in recovery, and the hæmophilic tendency is but rarely seen.

On the whole, therefore, it is evident that though splenic anæmia is a rare disease, its diagnosis is not generally one of any great difficulty.

Treatment.—The treatment must be symptomatic, and does not differ from that of other profound anæmias. All the usual remedies have been tried one after another, but so far nothing has been found to check the progress of the disease.

SAMUEL WEST.

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HÆMOPHILIA

Definition.—By hæmophilia we mean a disease congenital and hereditary, marked by a tendency to immoderate bleeding on slight causes, lasting throughout the life of the patient; and further accompanied by a troublesome tendency to a joint affection, which is often as wearisome to the patient, as the tendency to external hæmorrhages is dangerous.

The name of hæmophilia is modern, and seems to have been first introduced by Schönlein about 1828. "Hemophil" is, however, the name of one of the *dramatis personæ* in John Ford's *Broken Heart*, published in 1633. The disease was not classified or described until the time of Schönlein, though single observations may be found scattered in medical literature, beginning with the Spanish Albucasis or Alsaharavius in the 11th or 12th Christian century. The men who are the subjects of hæmophilia are called "bleeders," a word which appears early in the nineteenth century in the medical literature of the United States of America.

Etiology. — Beyond hereditary transmission hardly anything is known of the causation of hæmophilia. It affects especially the male sex. Though cases in women have been described, it has never fallen to my lot to see a definite case in a woman, yet the women in the bleeder families pass on the disease to their male offspring.

The mode of the hereditary transmission of hæmophilia is noteworthy. In a bleeder family we commonly find all the women free from the disease, while their brothers suffer. Then these women, if they marry and are fertile, bear a family some or all of the boys of which are bleeders, while all the girls escape. But these girls, if they marry, pass the disease on to their sons; and so the disease is continued.

This mode of hereditary transmission is not particular to hæmophilia. It is very well marked in colour-blindness; and in 1881 I published a genealogical tree showing the descent of this infirmity in a family since 1684. It is also seen in polydipsia, another congenital disorder, as Dr. Gee has pointed out; and the same mode of transmission may be seen now and then in ichthyosis, in the pseudo-hypertrophous paralysis of Duchenne, and in gout. The disease does not seem to descend from father to son; nor through the sons of a bleeder family, free from the disease, to their offspring. As a general rule, the sons of bleeders, and the sons of the brothers of a bleeder, who are free from hæmophilia, show no signs of the disease. But the daughters of a bleeder, like his sisters, pass on the disease to their male offspring; the daughters' offspring often showing the disorder in a highly aggravated form.

There seems at this moment no evidence that the marriage of near kinsfolk causes the disease. Nor is hæmophilia peculiar to any race of men. I think, however, that it is found more often amongst Jews, in proportion to the population, than amongst Englishmen. Cases have been described amongst the aborigines of Sumatra; and I notice that Japanese writers have lately begun to describe the disease.

No class in life seems to be exempt from hæmophilia; nor can any particular geographical distribution be assigned to it.

Symptoms.—As a rule the symptoms of hæmophilia appear during childhood; in the large majority of cases before the tenth year. In May 1884, at St. Bartholomew's Hospital, I myself saw a case amongst Dr. Andrew's patients in which no symptoms had been noticed until the lad was 19 years old. Other instances have been recorded in which the first symptoms were seen at the age of 21. But the absence of all early symptoms of hæmophilia can seldom be proved.

Hæmophilia may even show itself in fœtal life. An eight months' fœtus, from a bleeder family, has been found with bruising on the brow: and a boy when born had large extravasations over both shoulders. Severe hæmorrhage may take place after ritual circumcision, which is usually performed on the eighth day after birth; but as a rule the hæmorrhagic disposition does not show itself until near the end of the first twelve months of life.

There is a rare affection, one of the chief symptoms of which is a hæmorrhage on the falling off of the umbilical cord. This hæmorrhage rarely occurs in children of bleeder families; and it is separated from hæmophilia by the fact that when the child recovers it shows no further disposition to hæmorrhage in after-life (*vide* p. 561).

The joint troubles, which are so prominent in definite cases of hæmophilia, may appear early in life. In one of Brigstocke's cases an ankle became distended by effusion a few days after birth; but as a rule the joints do not begin to give trouble until the patient be four or five years of age.

The boys who are the subjects of hæmophilia have no external peculiarities. There is nothing constant about their complexion, stature, or muscular strength; nor can it be said that they are always intelligent, or that they distinguish themselves at school. Physical examination detects nothing amiss with chest or belly; neither spleen nor liver is enlarged, and the urine, in the cases in which I have examined it, showed the ordinary percentage of urea, and no decided alteration in its other constituents.

Certain states of the surroundings have been supposed to excite hæmorrhages in a bleeder, or to aggravate his hæmorrhagic disposition. Such are the difference in the seasons, day and night, cold and heat, changes of the moon, and the like; but these assertions greatly lack confirmation. Some have thought that the use of wine provokes hæmorrhages; others that the hæmorrhagic disposition is increased after the first traumatic hæmorrhage. This sequence, of course, admits of another explanation. Anger and other emotions have also been accused. There can be no doubt, however, that the disposition to hæmorrhages in these patients varies very much; and the observer must be very cautious in drawing conclusions as to the action of remedies.

The positive symptoms of hæmophilia may be divided into three heads: spontaneous bleedings, traumatic bleedings, and the joint affections.

The spontaneous bleedings are said by some observers to be oftentimes preceded by distinct *prodroma*. I have never been fortunate enough to observe these prodroma, but they are set out at length by Wachsmuth. They occur three or four days before the onset of the bleeding, and are mainly signs of plethora: the face is full, the lips and ears swollen, red, and hot; or the friends remark that the patient is looking remarkably well.

As might be expected, epistaxis is the most common of the spontaneous hæmorrhages, especially in childhood. Few bleeders live to any age without suffering from some form of nose-bleeding. Nor does it favour any of the ancient beliefs by flowing more from one nostril than the other. Bleeding from some part of the mouth comes next in frequency, though far behind.

As puberty comes on, hæmaturia and bleedings from the bowels replace the hæmorrhages from the nose and mouth. Very rarely spontaneous bleedings are seen from the conjunctiva, the skin, the ears, or the ends of the fingers.

The amount of blood lost in this way may be very trifling, or it may be so great as to kill the patient. There may be but a trifling ecchymosis or petechia under the skin, or the whole of the blood may seem to leave the body. Of the bleedings from the mucous membranes epistaxis is the most frequent cause of death; next to that, bleeding from the mouth, bowel, or lung. Hæmaturia is but rarely mortal.

On the whole, the traumatic hæmorrhages are the most to be dreaded. A mere scratch is sometimes sufficient to put life in danger. Trifling injuries, like the scarifications necessary for vaccination, the division of the frænum of the tongue, the application of a leech, or ritual circumcision, have caused death. The taking out of a tooth is an exceedingly dangerous act; it is a common cause of death in a bleeder, and should never be undertaken in a patient of this kind.

These patients vary, however, very much in the amount of injury that they bear. At one time the same patient will endure injuries the infliction of which at another time will endanger life; and in some families of bleeders the traumatic hæmorrhages are, as a rule, nothing like so dangerous as in others. I have myself seen a tooth drawn in such an one without any remarkable hæmorrhage; but, as a rule, this little operation is in the highest degree dangerous. Usually the vaccination of a bleeder is followed by no ill effects; this is also the experience gained by the practitioners in the canton of Graubünden, where there are many families of bleeders.

The opening of abscesses, either by the surgeon's knife or of themselves, is usually followed by a profuse hæmorrhage. This is still more profuse and dangerous when blood tumours or extravasations of blood are opened.

Death may come on very rapidly, within a few hours; or the bleeding may last for weeks. Often the uncontrollable hæmorrhage does not come on with the first infliction of the wound, but some hours or even a day after the hurt has been given. The quantity of blood lost in a few hours may be enormous.

Of the composition of the blood thus lost we have no very recent or exact observations. That first poured out seems to clot naturally, but after a great deal has been lost, it coagulates feebly or not at all. After great losses, it looks like water in which meat has been washed, and hardly stains the linen. With the microscope a certain increase in the white corpuscles has been noticed after hæmorrhages, as might be expected.

Symptoms of true anæmia follow the great loss of blood, and the patient dies bloodless. If, however, the result is to be favourable, the patient lies long as in a deep sleep, and on awaking he suffers for weeks and months from pallor and general bloodlessness. The blood lost is very slowly regained. During convalescence a depraved appetite for sand, chalk, and the like has been noticed in some few cases.

It has been said already that in hæmophilia the affection of the joints is more troublesome to the patient than the attacks of external hæmorrhage. The joints become swollen and painful, and are apparently filled with fluid; this disorder is continually recurring, so that some patients are rarely free from it, and they become cripples from the state of the knee. The knee is the joint which suffers the most; after that comes the ankle, then the elbow, shoulder, and hip joint. The wrist and the joints of the fingers and toes are seldom affected. When the acute swelling is over, the joint may recover completely for this turn; or some impairment of motion may be left; indeed after many attacks the joint at last may be almost destroyed.

The cause of this swelling is, in my opinion, an effusion of blood into the joint. Formerly it was thought that its origin was sometimes rheumatic, but it has been now shown from the necropsies of some cases that the joints contain blood; and this seems the most likely cause of the phenomena in all. Allied are, no doubt, the joint troubles seen in some cases of purpura which are not rheumatic.

Morbid anatomy.—Morbid anatomy has hitherto given a negative result in hæmophilia. As a rule, nothing but great bloodlessness can be found; and a good number of necropsies have now been made by trustworthy observers with this as the only result. Schönlein seems to have laid great weight upon certain changes in the heart, the deficiency of the muscular fibres in the walls of the septum; but he may have been describing only the "unprotected spot" of Peacock. Others have described a thinness of the walls of the arteries, but this appearance has been found in too few cases to justify us in regarding it as a constant element of hæmophilia.

Nor has the microscope anything to tell us in hæmophilia. Dr. Klein examined for me with the microscope several cases dying in St. Bartholomew's Hospital; but in no case was he able to detect any disease in the vessels or tissues. Dr. Percy Kidd observed certain changes in one case; but they have not been found in others, and it may be concluded that they were accidental and not essential to hæmophilia.

Of the *pathogeny* of a disease like hæmophilia it is really useless to speak. It would be mere speculation, and in a work like the present it would be undesirable to take up space with a discussion of mere opinions.

On the morbid anatomy of the joints in hæmophilia much light has been thrown of late years. It would seem that the repeated hæmorrhages into the joints lead up to a state which cannot be distinguished from chronic rheumatoid arthritis. The first blood effused into the joint seems to be reabsorbed, leaving the cartilages free from change. But as hæmorrhage succeeds hæmorrhage deep changes make their appearance. First the cartilages and synovial membrane are slightly coloured from the presence of blood, but the cartilages remain shining and smooth, and show no further change. In a further stage the cartilages become deeper coloured, of the rusty colour so often seen as the result of blood-staining, and they lose their clear appearance and become clouded and thin. Next fibrous bands pass from the femur to the tibia, the cartilages are rough and greatly thinned so that the bone is almost laid bare, and under the microscope changes very like those observed in the cartilage of chronic rheumatoid arthritis are observed. There are some specimens of this state of the joints in the Museum of St. Bartholomew's Hospital (740 *a. b. c. d.*).

Diagnosis.—When a boy, born in a bleeder family, begins to suffer from petechiæ, suspicion is at once aroused; and if, later, repeated nose-bleeding, hæmaturia, and joint troubles appear, the diagnosis is rendered highly probable; if to these be added a long-continued hæmorrhage after slight wounds, there hardly remains room for uncertainty.

Difficulty, however, often arises in practice when a patient presents himself of whose history little or nothing is known, and the trustworthiness of whose statements is somewhat doubtful. In the first place the sex is of the greatest importance. I have said that I have never seen a case of true hæmophilia in a woman, and I am inclined to think that the diagnosis of cases of hæmophilia in women is founded on mistaken observations. Next in importance are uncontrollable hæmorrhages after wounds, especially slight wounds, or wounds that ordinarily give rise to little hæmorrhage; for example, the taking out of a tooth is often a touchstone in the diagnosis. If no hæmorrhage have followed this operation, the opinion against the diagnosis of hæmophilia is materially strengthened. Then the joint affection in persons already suspected of hæmophilia adds something in favour of the diagnosis, though it must be remembered that temporary hæmorrhagic diatheses, like scurvy and purpura, sometimes show a joint affection which appears to be due to hæmorrhages into the joint affected.

In some doubtful cases, especially in children of a bleeder family, it may be well to wait a few months or even a year till more decided symptoms show themselves, as nose-bleeding and the like, before giving an opinion.

It should always be kept in mind that hæmophilia is a congenital general disease, which persists throughout the life of the patient. Temporary hæmorrhagic diatheses, like scurvy and purpura, must not be accounted hæmophilia; nor should a long-continued hæmorrhage from one single part have this name given to it, as a German writer, who ought to know better, has lately done.

There are certain rare cases with umbilical hæmorrhage in infants which may be confused with hæmophilia. They have profuse bleeding from the place of separation at the navel, they are often jaundiced, and they die after having shown signs of a general hæmorrhagic disposition. Such cases of umbilical hæmorrhage, however, do not belong to hæmophilia. They occur in children of both sexes, and not especially in bleeder families; and in those which survive, no tendency to hæmorrhage is noted [*vide* following article].

Prognosis.—The remote prognosis of hæmophilia no longer appears so serious as was formerly supposed. In Grandidier's figures only 10 per cent attained the age of 21. My own direct experience has been much more favourable. I have watched the boys of many bleeder families from childhood; and they have grown up into manhood, not without pain and suffering it is true, but they have kept their life, and followed easy occupations by which they gained their bread in a fashion. The patients whom I have thus watched have belonged mainly to the lower middle classes. They have known of their tendency to hæmorrhage, and thus in many instances, no doubt, they have been able to ward off wounds. With sufficient care, middle age would seem to be within the reach of many of them.

It is said that with the approach of middle age the tendency to hæmorrhage may disappear. I have never seen such a case; but the fact that it has been noticed by some observers should be remembered.

Treatment.—First of all it may be well to consider the measures to be avoided. All procedures by which blood is drawn—blood-letting, leeches, lancing of gums, scarifications and the like—must be forbidden. The red-hot iron and even blisters are attended by considerable danger in many cases. Surgeons should be warned that when the great operations of surgery have been performed, such as an artery tied in its course, amputation of a member, or lithotomy, the patient has usually bled to death of the wound. It is hard to imagine a case in which the use of the knife would be justifiable.

With traumatic hæmorrhages, it would seem best to follow the ordinary rules of surgery, always remembering the warning given in the paragraph above. Styptics, it may be noted, are generally worthless. The use of the hot iron and of the perchloride of iron is specially to be avoided.

In like manner, spontaneous hæmorrhages should be treated according to the general rules of medicine. Ice may be passed up the nose in epistaxis, or into the bowel in bleeding from the rectum, or placed in the mouth in bleeding from the gums. Whatever may be done will, however, too often prove ineffectual; and if the medical attendant have courage enough for such a line of action, there would almost seem to be a better chance for the patient if the attendant abstained altogether from active local treatment designed to check the bleeding. When all the blood seems to have left the body, and the patient about to die of the loss of blood, it has not infrequently happened that the hæmorrhage has ceased and the patient has slowly and gradually recovered.

Transfusion of blood as a last resort has been practised in some cases with success; and in desperate cases I should feel inclined myself to recommend its employment.

During the intervals between the attacks of hæmorrhage, what shall be done? The hygienic treatment is of great importance; all occasions of hæmorrhage must be warded off; all persons about the boys should be told of the tendency to hæmorrhage, and of the grave consequences which may follow the slightest injury. Nearly all the ordinary games of boys, amongst which are specially to be named cricket, hockey, and football, have to be forbidden. Other sports and exercises may be allowed according to their results.

Cold sponge bathing is useful and well borne. The dwelling-place should be dry, the air bracing; during the winter some have seen good from a removal to a dry and warm climate like that of the Riviera. It is to be regretted that we have so little experience of climatic treatment. Warm clothing seems very desirable, as these patients often feel the cold severely.

As to drugs, a good deal of caution must be exercised in drawing conclusions even from an experience which may seem to be wide. For instance, at the beginning of this century, the American physicians who had treated families of bleeder children tell us that "the sulphate of soda was accidentally found to be completely curative of the hæmorrhage" in hæmophilia. At the present day no such great confidence is felt in the sulphate of soda. At this moment the chlorides are in greater favour; the chloride of calcium has been recommended in these cases from its supposed power of increasing the coagulability of the blood, and very favourable results have been reported from its use. I cannot, however, say that I have seen anything like a complete disappearance of the hæmorrhagic disposition follow even a long-continued use of this drug.

In my own experience I have found very good results follow a course of cod-liver oil and perchloride of iron alternately.

In the treatment of the acute stage of the joint affections, rest is the very first, almost the only element in the cure. The joint must be rendered motionless, as soon as the patient can bear such treatment, by splints of plaster of Paris or whatever the surgeon may deem suitable. Pain must be neutralised by opiates; and there does not seem much danger in hæmophilia from subcutaneous injection of morphia.

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HÆMORRHAGES IN NEW-BORN CHILDREN

THE hæmorrhages which occur in new-born children may be divided into two groups: (1) **Traumatic or Accidental Hæmorrhages**, which are the direct result of injury at the time of birth; and (2) **Spontaneous Hæmorrhages**, which occur without any apparent external cause.

The cases of spontaneous hæmorrhage are best again subdivided into (*a*) *Idiopathic* cases, where the bleeding is the chief or only symptom—the so-called "hæmorrhagic disease of new-born children"; and (*b*) *Symptomatic* cases, where the hæmorrhages are secondary to some serious organic disease, such as congenital malformation of the heart, congenital obliteration of the bile-ducts, or some grave affection of the liver. With this group also may be taken the rare cases in which true hæmophilia leads to hæmorrhages in early infancy.

I. TRAUMATIC OR ACCIDENTAL HÆMORRHAGES

The traumatic hæmorrhages are mainly due to injuries received during birth, either by the pressure of the maternal parts on the child, or by the artificial means used by the accoucheur to expedite delivery. They are consequently more frequent in the case of first-born and male children, in difficult and prolonged labours, and when the presentation is abnormal. There can be little doubt, also, that increase of the blood-pressure, owing either to asphyxia from pressure on the cord, or to pressure on veins or compression of some other part of the body, may be an important cause of their occurrence.

Traumatic cases are of less importance, from the physician's point of view, than those of spontaneous bleeding. Nevertheless they also are of interest to him, and therefore worthy of brief mention here because, in not a few instances, they form the starting-point of serious nervous disease in later life.

The most important situations in which the effused blood is found may be stated as follows:—(i.) On the surface of the skull, between the pericranium and the bone—cephalhæmatoma; (ii.) Inside the cranium—apoplexia neonatorum; (iii.) Into the substance of the sterno-mastoid muscle; (iv.) Into one or more of the abdominal or thoracic organs.

Cephalhæmatoma

Cephalhæmatoma is the name given to a swelling on the surface of the cranium formed by a collection of fluid blood between the pericranium and the bone. The condition is due to rupture of blood-vessels under the pericranium, owing to mechanical pressure during birth; and it is met with about once in every 200 births. It is much more commonly seen after first labours than after subsequent ones, and is especially frequent after difficult births in which the head has presented. It occurs, however, sometimes after breech cases, and occasionally also with comparatively easy and normal labours.

In the majority of cases the tumour is situated over the right parietal bone—this being usually the presenting part. Less frequently it is found in the left parietal region; and sometimes it occurs on both sides. It is rarely met with over the other cranial bones. The swelling is generally noticed within the first two or three days after birth. It is rounded in contour, fluctuating and not apparently tender; and it shows no heat or redness. Being under the pericranium it is always distinctly limited to the surface of one bone and never crosses a suture. For four or five days it usually goes on increasing in size, and then, after being stationary for a while, it slowly disappears. When the blood begins to be absorbed, the bone can readily be felt through the tumour, and round its margin a raised ring can be made out. This is due to the formation of bone having gone on under the raised periosteum. Sometimes also from the same cause there is a crackling sensation experienced on handling the surface of the swelling.

Generally the blood tumour is quite absorbed within four weeks of birth, but it often takes two or three months before all trace of the bony ridge round it has disappeared. The prognosis in uncomplicated cases is invariably good, the cephalhæmatoma always recovering without any treatment. It should, however, be remembered that in a certain proportion of cases the external swelling is accompanied by an intracranial hæmorrhage.

Intracranial Hæmorrhages (Apoplexia Neonatorum)

Intracranial hæmorrhages are unfortunately not very uncommon. They are important, not only because they are a very frequent cause of still-birth and early death, but also because they are responsible for a large amount of bodily and mental defect in after-life.

Etiology.—Although the state of asphyxia into which many children are brought during birth must be regarded as a strongly predisposing element in the causation of these hæmorrhages (Ashby), Spencer's statistics render it almost certain that they are mainly due to external injuries. These may be produced either by the pressure of the maternal parts on the child or by that of the blades of the forceps. Thus he has found that the frequency of cerebral hæmorrhage is greatest with forceps delivery, less when the breech or foot presents, and least of all with natural head delivery.

As to the mechanism by which the lesion is brought about, Spencer suggests that in many cases it is due to displacement inwards of the lower anterior corner of the parietal bone. This corner directly overlies the great anastomatic vein, and being driven inwards during birth, clamps this vessel so as to cause engorgement in its area of distribution. This explains, he thinks, the fact that the hæmorrhages are often limited to the parietal region and Sylvian fissure. Compression of the internal jugular vein by the forceps may also favour cerebral hæmorrhage in a similar way. S. McNutt has shown that hæmorrhages limited to the convexity are more frequent in breech than in head deliveries.

Morbid anatomy.—In a very large majority of the cases the hæmorrhage is primarily meningeal, and the injury to the brain itself is secondary and results from pressure from the surface. In some cases, however, the bleeding takes place into the brain substance. Effusion of blood on the inner aspect of the cranial bones, outside the dura mater (internal cephalhæmatoma), is sometimes found along with an external cephalhæmatoma. It is said that this does not occur unless a fracture of the bone is present (Holt). Generally the blood is poured out into and beneath the arachnoid and pia.

The lesion is more frequently bilateral than confined to one side; and it is much commoner at the base than on the convexity of the brain. It is also commoner towards the posterior part of the skull than anteriorly. As has been already mentioned, hæmorrhages are frequently found over the parietal region and in the Sylvian fissure. The ventricles are sometimes distended with blood.

When meningeal apoplexy occurs, it sometimes lacerates the underlying cerebral cortex; in any case, it is apt to interfere with its nourishment by tearing through the blood-vessels which pass into it from the membranes. It also injures it by its pressure, so that softening and atrophy soon follow. Thus permanent atrophy and arrest of development of the cortex are set up along with degeneration of the fibres in the motor tract of the brain and spinal cord.

Clinical features.—If the damage to the brain be very great, the child will probably be still-born. The hæmorrhage may, however, be tolerably extensive, and yet the infant may survive for several days; or it may even recover and grow to adult age, although with a permanently damaged nervous system.

In many cases where the hæmorrhage has been severe the infant does not at first show any distinctively cerebral symptom, except torpor and feeble or irregular breathing; but other indications may be present. There may be obvious paralysis of one or more limbs, or of the cranial nerves if the hæmorrhage implicate the base. If the child live for some days, convulsions often occur; and they are more frequent in cases where the hæmorrhage is over the cortex than in those where it is at the base of the brain (McNutt).

As the child gets older, although the parents are often slow to see that anything is the matter with him, it will usually soon be found that the limbs are unnaturally stiff and the knee-jerks exaggerated. Later, he is backward in learning to hold his head up, in sitting up, and in walking; or it may be that he displays a lack of interest in his surroundings, which is soon noticed as abnormal. Gradually, as the brain grows, the extent of the damage to its functions becomes more manifest, and the case passes off into spastic paraplegia, hemiplegia, diplegia, imbecility, or idiocy with or without paralysis.

Prognosis.—While extensive hæmorrhages at the base are usually fatal, comparatively large ones over the convexity are compatible with life. Small cortical apoplexies may, it is said, be entirely recovered from, but in the great majority of cases more or less permanent injury to the brain results.

Hæmatoma of the Sterno-mastoid (Sterno-mastoid Tumour)

Hæmatoma of the sterno-mastoid is a collection of blood which forms within the sheath of that muscle when some of its fibres have been ruptured during birth. It is met with most frequently in breech cases or cross-births where there has been difficulty in delivering the head; often also in difficult forceps cases, and sometimes after labours which are said to have been quite easy. It is probably caused more often by a sudden twisting of the neck than by simple traction on it. In nearly 300 autopsies on children, who were either still-born or had died soon after birth, Dr. Spencer found this lesion in fifteen.

The swelling may occur at any part of the muscle, but most frequently it is situated about its centre. The muscle of the right side is much oftener affected than that of the left. The tumour is not usually noticed until the second or third week, and often it does not attract attention till much later. This is due to the fact that the swelling caused by the effusion of blood is generally small at first. It is not until the injury to the muscle has occasioned a considerable growth of fibrous tissue ("muscle callus") round it, that it becomes too obvious to be overlooked. It may reach the size of a pigeon's egg, but it is generally smaller. The swelling remains stationary for weeks, and then slowly diminishes. It usually takes six or seven months to disappear; but it may have quite gone by the third month, or it may last more than a year (Pollard).

The connection between hæmatoma of the sterno-mastoid and so-called congenital wryneck is certainly a very close one, but it is difficult at present accurately to define it. Out of 106 cases of hæmatoma which Mr. D'Arcy Power collected from medical records, marked wryneck had resulted in twenty-one at least, while only in fourteen had it been specially looked for and not found. Dieffenbach and other older writers on the subject assumed that the wryneck was the simple result of the injury to the sterno-mastoid muscle at birth; but this explanation has of recent years been much disputed (Golding-Bird, Petersen, Koettnitz). The chief difficulty in accepting it lies in the fact that in most if not all of the marked cases of congenital wryneck there is a very decided arrest of growth of all the structures of the face on the affected side, and it is difficult to imagine how any lesion of the neck only could bring this about. What cerebral lesion could cause it, however, is as yet undetermined. The fact remains that this characteristic facial asymmetry along with wryneck not uncommonly appears as a sequel of hæmatoma of the sterno-mastoid (I have recently watched this sequence in three instances). Usually little can be effected in the way of treatment owing to the age of the child; but it is probable that judiciously applied massage and manipulations may sometimes be of use.

Hæmorrhages into Abdominal and Thoracic Viscera

As Dr. Spencer points out in his valuable paper, hæmorrhages into the abdominal and thoracic viscera are much more frequent after breech cases than after those in which the head has presented. They may occur into any of the organs, being seen frequently in the lungs, liver, kidneys, suprarenals, and intestine, and comparatively rarely in the spleen.

In most cases of internal hæmorrhage the diagnosis is impossible during life for lack of symptoms. Pulmonary infarctions, however, sometimes betray their presence by causing physical signs of consolidation of the lung. They are the cause of death in many cases of children who live for a few days only, and in these the fatal issue is apt to be attributed to congenital heart disease on account of the degree of cyanosis which is present. If the infant live long enough, pneumonia may result.

Hæmorrhage into the pyramids of the kidneys may cause death within a few days, with symptoms of suppression of urine; and Spencer records one case in which a large hæmorrhage into the cæcum caused complete intestinal obstruction.

II. SPONTANEOUS HÆMORRHAGES

(*a*) Idiopathic Cases (the Hæmorrhagic Disease of New-born Children)

Description.—The hæmorrhagic disease of new-born children may be described as a passing morbid condition of the system which shows itself mainly by a tendency to spontaneous bleeding. The hæmorrhages may occur either from the umbilicus, from the stomach or bowel, from the blood-vessels in the subcutaneous tissue, or from other parts; and their occurrence is probably due to poisoning by the toxin produced by any one of a variety of micro-organisms.

The condition is a very rare one. Cases of gastro-intestinal hæmorrhage, which is its commonest form, are said to occur once for every 500-1000 births (Runge, Hermary, Kling); while umbilical hæmorrhage is only met with once for every 5000 confinements (Ribemont).

Clinical features.—In the great majority of cases no family history of bleeding is to be obtained. The sexes seem to be affected in about equal proportions. This point is of interest when we remember the very much greater frequency with which the male sex is affected in hæmophilia—the proportion being stated by Grandidier as eleven boys to one girl (*vide* art. "Hæmophilia," p. 549).

Sometimes the patient is in weak health before the bleeding sets in; being premature or feeble, or perhaps the subject of congenital syphilis. Generally, however, he appears quite healthy until the hæmorrhage begins. This takes place usually within the first week of life, and rarely after the end of the second; the exact date varies, partly according to the situation whence it comes.

The site of the hæmorrhage may vary considerably. In the majority of cases it comes either from the alimentary tract (mouth, stomach, or bowel), or from the umbilicus. It may also take place into the subcutaneous tissue, or from the nose, conjunctiva, or ears, or into almost any of the internal organs. The bleeding may come from one situation only, as often happens in the slighter cases, or many parts may be affected, either at one time or successively. Thus, of 50 cases reported by Townsend, the umbilicus was affected in 18 (alone in 3); the intestine in 20; the mouth in 14; the stomach in 14; the nose in 12: subcutaneous ecchymoses occurred in 21; bleeding from an abrasion of the skin in 1; from the meninges in 4; cephalhæmatomata appeared in 3; hæmorrhages into the abdomen in 2, and into the pleura, lungs, and thymus in 1 each.

The amount of blood lost at a time is usually small; but the loss is generally so frequently repeated that pallor, chilliness, and prostration with failure of the pulse are very soon produced. In some cases the temperature is high, in others it is normal or subnormal throughout. In the cases of recovery the bleeding usually lasts one or two days; in the fatal cases death often occurs within twenty-four hours, and rarely later than three days from the beginning. Sometimes diarrhœa appears, and towards the end convulsions not infrequently set in.

In children who recover the convalescence is apt to be prolonged and tedious, although the health is not permanently damaged.

Gastro-intestinal hæmorrhages (*Melæna neonatorum*).—The blood in these cases is more frequently passed by the bowel than vomited. When vomited it is sometimes bright red, often dark brown in colour. Its amount varies greatly in different cases. Sometimes the hæmatemesis occurs only on one occasion; more frequently small quantities are brought up repeatedly. When passed by the bowel the blood is generally very black and thoroughly mixed with the motions. Sometimes, when it comes from the lower part of the bowel, it is red, and it may be in clots.

The blood is most frequently seen for the first time on the second day of life, or at least before the fifth; but occasionally the hæmorrhage may begin as late as the second week.

Spontaneous umbilical hæmorrhage usually takes the form of a steady oozing from the point where the cord has separated or is about to separate. The bleeding does not generally come from any visible blood-vessel; it is often intermittent; sometimes it is alarmingly free. Sometimes it takes place into the substance of the cord, or from fissures on its surface.

It generally begins about the fifth day of life, but it may occur earlier, and it may be deferred till the seventh or even the ninth day. It does not generally last more than three days, but in rare cases it may go on much longer. It is often fatal within twenty-four hours.

Subcutaneous ecchymoses may occur at any part of the body, and are as common on protected areas as on those which are exposed to pressure and friction. They are often of small size, but occasionally they become very large. If they occur without any hæmorrhages from other situations the prognosis is generally good.

Bleeding from the female genitals occurs occasionally in cases of multiple hæmorrhages. It is, however, much more frequently met with as an isolated symptom; and, when this is so, it is generally the result of some trifling local disturbance and has no serious significance. The hæmorrhage begins usually within the first six days of life, rarely after the twenty-first (Busey). The external genitals appear quite normal, but there is a more or less constant oozing of blood from the vaginal orifice, which lasts from two to five days, or sometimes a little longer. Owing to the trivial nature of the ailment very few opportunities have occurred for investigating its morbid anatomy. Billard asserts that the blood comes from the uterine mucous membrane, and Eröss in one case found acute hæmorrhagic catarrh of the fundus uteri. Only in rare cases does it recur, so that it cannot be regarded as of the nature of menstruation.

Morbid anatomy.—In most cases of children who have died from hæmorrhage there is nothing to be discovered at the autopsy but the traces of the effused blood and a general pallor of all the organs.

In a considerable proportion, however, of the cases of melæna (40 per cent according to Romme), more or less ulceration of the mucous membrane of the stomach or duodenum has been found. The ulcers are usually multiple, and may either consist of superficial abrasions or be of a perforating character. In one case (Landau) a clot was found obliterating the blood-vessels which supplied the area in which the ulcers were situated, but this is not usually the case. In some cases of melæna cerebral hæmorrhages have been found (Pomorski and v. Preuschen), but these also are by no means constant. In syphilitic cases endarteritis of the small and middle-sized vessels in the submucous tissue of the alimentary tract has been described (Mracek).

A considerable variety of micro-organisms has been found in the blood and in the tissues in cases of hæmorrhage, and especially in those of melæna. The first to record an observation of this kind was Klebs, who, in 1875, described a micrococcus which he had found in large quantities in the organs of nine new-born children who had died of hæmorrhage. This he injected into young rabbits, and succeeded in producing hæmorrhages in them. His results were confirmed in the following years by Weigert, Eppinger, and Rehn. Since then various micro-organisms have been discovered in these cases by competent observers. Thus, streptococci have been found by Baginsky, Babes, and Bar; bacillus pyocyaneus and staphylococci of various kinds by Neumann, Bar, and Schäffer, and bacterium lactis aerogenes by Neumann and Schäffer. Further, Babes found in one case an organism with all the characters of the diplococcus pneumoniæ, and Dungern one which resembled in every way Friedländer's pneumococcus. In Dungern's case it is also recorded that, while the child was in the ward, three other infants died of severe pneumonia.

In 1894 Gärtner published an account of two fatal cases of melæna in which he found a short bacillus. Cultures of this organism were made and injected into the peritoneal cavity of young puppies, and they set up fatal gastro-intestinal hæmorrhage. In one of Holt's cases a similar organism was found.

Etiology.—A number of very different hypotheses of the causation of this condition have been framed. The following deserve mention:—

1. Von Preuschen and Pomorski have published cases where melæna and pulmonary infarction of an apparently spontaneous origin were found after death to be associated with traumatic hæmorrhages into the cerebral peduncles and the fourth ventricle, damaging the vaso-motor centre. They therefore maintain that many if not all of the cases of spontaneous hæmorrhage are secondary to cerebral injury. They were able to strengthen their position by means of experiments on animals; for they succeeded in producing melæna in a considerable number of rabbits by puncturing the cerebral peduncles and the walls of the fourth ventricle.

While these observations are certainly interesting and important, they cannot be held as explaining the occurrence of most cases of this disease. Cerebral hæmorrhages have only been found in a few instances.

2. Other writers have laid great stress on the local morbid condition. Thus, for example, in dealing with melæna they have given mechanical explanations to account for the presence of ulceration in the stomach and bowel in these cases. The most remarkable of these hypotheses is that put forward by Landau. He noticed that the condition often occurred in premature and weakly infants in whom the function of respiration was established with some delay and difficulty. He accounts for this by supposing that the delayed inspiration favours stagnation and clotting of the blood in the umbilical vein. Then, he further supposes that, from the thrombus so formed or from that in the ductus arteriosus, an embolus is separated and carried through the circulation until it becomes impacted in one of the arterial branches which supply the stomach and duodenum, and ulceration results. In one case of gastric hæmorrhage he was able to satisfy himself that the artery supplying the area from which the blood came contained a clot.

Emboli of this sort have not been found by other observers who have looked for them, and Landau's theory has not, therefore, been generally accepted. It must be remembered in this connection that the formation of ulcers in the stomach and bowel is a frequent result of general infection with organisms of various kinds, and even of poisoning by toxins (Demelin).

3. Considerable stress has been laid by some authors on the fact that many of the patients in these cases are syphilitic or otherwise weakly; and it has been supposed that the bleeding might be attributed to some disease causing special fragility of the blood-vessels.

Evidence of vascular disease, however, has not usually been found; and it seems more probable that debilitated states of the system act as remote causes only in so far as they prepare a suitable soil for the growth of micro-organisms.

4. Of recent years there has been an increasing tendency to regard the spontaneous hæmorrhages in these cases as a manifestation of a micro-organismal disease; and, although this view can scarcely as yet be said to be thoroughly established, there are a great many facts in favour of it. It is well known, for example, that many pathogenetic organisms have the property of producing a tendency to hæmorrhage. As already mentioned, a large number of different organisms, known and unknown, have been cultivated from the blood and tissues in these cases; and some of them have even been found to cause hæmorrhages when injected into animals. Further, the symptoms of the cases and their short course point to the disease being an infective one, as do also the facts that they are more frequently met with in hospital than in private practice, and that they have been known to occur as an epidemic.

Diagnosis.—Spurious melæna, that is, the vomiting or passing by the bowel of blood which the child has swallowed during birth, or has sucked from fissures in the mother's nipples, often causes needless anxiety if mistaken for this disease. It is more frequently met with than true hæmorrhage. Or the child may have epistaxis or hæmorrhage from an ulcer in the mouth or throat, and the blood may be passed with the motions and cause a diagnosis of melæna. Such mistakes are not usually difficult to avoid. If, however, the hæmorrhages be confined to the internal organs, they are very apt to be overlooked in the absence of characteristic symptoms.

The occurrence of spontaneous hæmorrhages—especially ecchymoses—has, it should be remembered, some interest from a medico-legal point of view, as they may be regarded erroneously as evidence of violence.

Prognosis.—The condition is always a very dangerous one. In Townsend's cases the mortality was 62 per cent. In cases of umbilical hæmorrhage it is even larger than this, being variously stated by authorities at from 65 to 84 per cent; while in melæna it is usually estimated at from 50 to 60 per cent. Should the infant be syphilitic or otherwise constitutionally feeble, this fact naturally renders the prognosis more unfavourable.

Treatment.—Great encouragement to prompt and persevering treatment of these cases is to be gathered from the fact that the disease is so brief in its duration. The treatment is to be conducted on general principles, and too much reliance is not to be placed on drugs. It is especially important that everything possible should be done to conserve the child's vitality. He should be kept perfectly quiet, and protected from cold by wrapping in cotton wool; he should also be surrounded, if necessary, with hot-water bottles. French writers recommend the use of a couveuse (Dusser, Oui). He should not be allowed to suck, but at short intervals by a spoon or medicine-dropper should have small quantities of his mother's milk, or diluted peptonised milk, cooled with ice. Small doses of ergotin may be given by the mouth, or, if the bleeding be severe, subcutaneously. If there be much collapse, it may be necessary to give alcohol by the mouth or ether as a hypodermic injection.

In melæna injections into the bowel are to be avoided. They are probably worse than useless, as they stimulate the intestinal movements. In umbilical hæmorrhage intelligent and patient digital pressure on the bleeding part is probably the best means of treatment. The actual cautery, nitrate of silver, and the application of plaster of Paris have also been successful in some cases. If other means fail, the base of the bleeding spot should be transfixed by a hare-lip pin and a ligature applied round it.

(*b*) Symptomatic Cases

Description.—Spontaneous hæmorrhages, similar in most respects to those we have been considering, are frequently met with as a symptom of various diseases. Thus we find them occasionally occurring in children with congenital malformation of the heart, rarely in infants who inherit true hæmophilia, and frequently in cases of congenital obliteration of the bile-ducts and other serious diseases of the liver accompanied by jaundice.

The tendency to hæmorrhage met with in these morbid conditions differs from that seen in the hæmorrhagic disease of new-born children in that it is permanent. With few exceptions it lasts as long as the child lives.

Children with congenital malformation of the heart do not often suffer from spontaneous bleeding; and although hæmophilia generally manifests itself for the first time in childhood, it is very rare indeed to find it as a cause of hæmorrhages as early as the first year of life. Out of 576 cases of hæmophilia, of which Grandidier collected details, the bleeding occurred in early infancy in 12 only.

In congenital obliteration of the bile-ducts, however, and in all other forms of disease which cause lasting jaundice in young infants, hæmorrhages are a common and characteristic symptom. Thus, more than two-fifths of the cases of umbilical hæmorrhage collected by Jenkins and Grandidier occurred in icteric infants; while in 65 cases of congenital narrowing or obliteration of the bile-ducts tabulated by myself, hæmorrhages were noted in more than half of the number of infants which had lived more than a few days. A similar hæmorrhagic tendency is of course well known to occur sooner or later in all cases of continued obstruction of the common duct; and is almost equally characteristic of a number of very different morbid conditions, all of which are accompanied by jaundice;

such as acute yellow atrophy, yellow fever, phosphorus poisoning, and so forth.

Clinical features.—The places from which the bleeding occurs in these cases are just the same as those observed in the case of idiopathic hæmorrhages. Their onset, however, is generally later. Thus in Grandidier's cases of umbilical hæmorrhage the average date of onset was about the sixth day in the non-icteric and about the tenth in the icteric cases. In the case of gastro-intestinal hæmorrhages this difference is very much more marked; for, although jaundiced infants sometimes show a tendency to hæmorrhage from the very first, they often do not begin to bleed until several months after birth. When once established the tendency seems rather to increase as they grow older.

Etiology.—The causation of the hæmorrhages in cases of jaundice has never been satisfactorily explained, although many hypotheses have been proposed to account for them. By some they have been attributed to impoverishment of the blood (Budd, Murchison); by others it has been supposed that they are due to bile acids circulating in it, and either acting on the corpuscles (Leyden) or setting up a diseased state of the blood-vessels (Wickham Legg).

It seems, however, more probable that the hæmorrhagic tendency is caused in some way by the presence in the blood, not of bile acids, but of ptomaines or some similar organic poisons. These are formed in the process of ordinary digestion, and the diseased liver is not able to render them innocuous, as it would do if it were in a state of health. The following facts seem to support this hypothesis. It has been found by Roger that the function of the liver, in virtue of which it neutralises the organic poisons formed in the alimentary canal, as well as others, is closely connected with the amount of glycogen it contains. Thus, when the liver contained little or no glycogen, he found that a very much smaller dose of these organic poisons was required to produce a given result than was necessary if the organ were healthy in this respect. It has also been demonstrated by Dr. Wickham Legg and others that the obliteration of the bile-ducts by ligature is followed in animals by disappearance of glycogen from the liver. It would appear that the retention of bile interferes with the proper discharge of the function of glycogenesis in the hepatic cells.

In the light of these observations, it seems not improbable that in congenital obliteration of the bile-ducts and other serious forms of jaundice a process of auto-intoxication is set up. If this be so, the poisons which come thus to circulate in the blood will probably induce hæmorrhages in the same way as do those toxins which are produced by the action of micro-organisms in the idiopathic cases.

Owing to the serious nature of the diseases present in these cases the **prognosis** is much worse than in the idiopathic group, and the **treatment**, which is to be conducted on the same lines as in the others, is even less likely to be successful.

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PURPURA

Definition.—Spontaneous extravasations of blood into the skin, mucous membranes, and internal organs of the body, sometimes accompanied by free hæmorrhages from mucous surfaces.

Etiology and Pathology.—Morbid anatomy simply reveals the existence and extent of distribution of hæmorrhagic effusions, often accompanied by evidences of anæmia. In a minority of cases in the mucous membranes, and more rarely in the skin, erosions or ulcerations are met with in connection with the hæmorrhages, but these are clearly the effects and not the cause of them; in mucous membranes the moisture of the part, and in some organs the digestive property of the secretions, tend to produce this result. In the hollow viscera blood may be found in considerable quantities, and the serous cavities may contain blood-stained serum. Besides the skin and mucous membrane, hæmorrhage occurs in the solid organs and in the serous membranes. They are found in the lungs, kidney, spleen, liver, brain, and retina; indeed, there is no part in which hæmorrhages may not occur. In the brain, from the delicacy of its structure and feeble resistance, the hæmorrhage may reach considerable magnitude, and may be fatal. The pleura, pericardium, peritoneum, and pia-arachnoid are often dotted over with small extravasations. The hæmorrhages vary in size from a pin's head to a patch as large as the palm of the hand. On post-mortem examination the most important changes found, other than hæmorrhages, are in the kidneys and lungs. Slight degrees of diffuse or parenchymatous nephritis are relatively common. Congestion and œdema of the lungs are frequently present, and are often the determining cause of death. Ulceration of the intestine and enlargement of the solitary and agminated glands are sometimes present.

With regard to the mode of escape of the blood in this as in other conditions in which spontaneous hæmorrhages take place, it may be by *rhexis*—by rupture of blood-vessels, or by *diapedesis*—by the escape of blood corpuscles through unbroken vessel walls. The former is most probably the process in the great majority of cases. Though many observers have failed to discover rupture of blood-vessels at the seat of the extravasations, Unna and his pupil Sack (20) have shown they are to be detected by certain methods of examination. According to Unna, it is the veins that give way; and he has pointed out that the laceration occurs especially at the junction of the superficial part of the subcutaneous tissue with the lower part of the cutis. At this point, which he regards as one of less resistance, the vessels lose their well-marked adventitia, and lack the support of the highly elastic cutis. The extravasated blood from its seat of origin percolates the epidermis, and occasionally the sebaceous and sweat glands; in some cases sero-hæmorrhagic extravasations take place also in the subcutaneous and intermuscular tissues. The causes that lead up to and actually determine the escape of blood are probably many and complex. Search has naturally been made in the walls of the blood-vessels for changes apt to cause them to give way. In some cases inflammatory changes have been found, and may in such instances have been the cause of the ruptures. In the majority of cases, however, the inflammation is the result of the violence to which the coats of the vessels have been subjected, an inflammation which may extend to vessels at some distance from the rupture. A hyaline degeneration, either of the intima or of the adventitia, or both, has been found by some observers. In the well-known case recorded by Wilson Fox a lardaceous change was found in the vessels of a syphilitic subject. Unna properly remarks, and experience of these changes in other circumstances confirms his opinion, that these hyaline and lardaceous changes would rather have a tendency to restrain than to encourage hæmorrhage. Venous thrombosis, as in so-called "purpura thrombotica," has been met with occasionally; but probably it stands in the relation of effect rather than of cause. Capillary emboli have been found in sarcoma (Hilton Fagge), in leucocythæmia, and in pyæmia, and may have a direct causal influence; but numerically such cases are very infrequent, and afford no explanation of the majority of cases of purpura in which they are absent. In recent years great attention has been devoted to the search for micro-organisms in the blood, in the blood-vessels, and in the tissues. Various bacteria have been found by different observers in some cases, but in other cases the same observers have failed to discover them. The presence of micro-organisms in the blood-vessels, even in large numbers as in diphtheria or anthrax, does not necessarily give rise to hæmorrhage; moreover, apart from the negative results of the search for bacteria, the circumstances in some cases in which purpura occurs make it unlikely that its causes are of this kind. Though thus not necessarily leading to rupture of vessels or diapedesis, they may nevertheless affect the vessel walls indirectly, by inducing some chemical change in them, as suggested by Watson Cheyne and Unna. Further, as Watson Cheyne has pointed out, the presence of bacteria does not necessarily imply that their entrance into the blood is the starting-point of the disease; the alternative view, however, may be entertained that, although the primary cause may be of quite a different nature, the result may be such an alteration of the fluids of the body that, of the innumerable organisms present in the mouth and intestinal tract, certain species may be enabled to penetrate into the blood and to live in it. It is quite possible, also, that some poisonous toxin or albumose formed in other parts of the body may be absorbed, and act chemically upon the blood-vessels, or on the vaso-motor nerves, producing variations of blood-pressure which at the weakest points they are unable to resist. In the whole class of specific diseases, whether in those in which micro-organisms have been demonstrated, or in those in which so far they are only assumed, the bacteria or their products must play an important part in the production of the cutaneous hæmorrhages which are an occasional feature of nearly all members of this group of diseases. The fact that purpuric phenomena are not uncommon in certain of them, such as scarlet fever and measles in which no specific micro-organisms have as yet been demonstrated, should make us chary of denying the possible existence of bacterial influence in the purpura of other diseases in which up to the present no micro-organisms have been found.

It is certain that cutaneous hæmorrhages are sometimes determined, and in all probability primarily caused by nervous influences; as in the case of purpura occurring in the situation of the lightning pains of tabes (Strauss, 19), and in connection with certain neuralgias (Weir Mitchell). The mechanism of the hæmorrhage in such cases is hitherto purely conjectural; but it seems most probable that, by acting on vaso-motor centres, it produces variations of vascular pressure under which the blood-vessels give way in the situation already indicated at the points of least resistance. Though purpura is one of the manifestations of hæmophilia, the histopathology of the latter need not be fully discussed here (see "Hæmophilia," p. 552), nor would it materially elucidate the pathology of the majority of cases of purpura. Hæmophilia is believed, however, by some authors to be due to a congenital defect in the vascular walls. It is quite possible that in some cases of purpura a hæmophilic taint may be an element in the hæmorrhagic tendency.

Venous stagnation plays a part in the production of purpura. Though not of itself a sufficient explanation of hæmorrhage, it is evidently a factor of importance, as in nearly all cases of purpura the hæmorrhages begin and are most marked in the lower extremities, the veins of which have to support a longer and heavier column of blood than those of other parts. As a rule, however, something more than stagnation is necessary to bring about rupture or diapedesis. When hyperæmia co-operates with stagnation the conditions are favourable to hæmorrhage (Unna).

Next, in relation to the escape of blood from the vessels, we have to consider the influence of the quality of the blood. The changes in the composition of the blood in purpura may be of the most varied kind: (i.) deficiency of the red corpuscles as in pernicious and other severe forms of anæmia, (ii.) excess of white corpuscles as in leucocythæmia, (iii.) deficiency or excess of some of the saline constituents of the blood as in scurvy, (iv.) alterations in the reaction, (v.) alterations in the specific gravity, (vi.) deficiency in the fibrin-forming elements may all play their parts in the initiation of changes in the vessel walls and in their permeability. Lastly, the presence in excess of some organic matters such as bile, urea, and other products of metabolic changes as in jaundice or uræmia, or the addition to the blood of extraneous matters, have all a tendency to promote some chemical or vital changes which render the vessels liable to rupture or increase the permeability of their coats. In the latter category we have important evidence of the effects of certain chemical substances. The observations of Prussak, confirmed by Wickham Legg, have demonstrated that chloride of sodium injected into the vessels or subcutaneous tissues of the frog gives rise to diapedesis of coloured corpuscles which, under the microscope, may be seen to pass through the intact walls of the blood-vessels (9). Similarly, in certain persons, iodide of potassium, as well as other drugs, give rise to purpura. Though the exact mode of operation of such agents has not been worked out, we must ascribe some influence, direct or indirect, to chemical action on the walls of the blood-vessels.

Finally, it must be pointed out that a diminution of support to the blood-vessels by the tissue immediately surrounding them may lead to their rupture. Thus purpura occurs in those who have wasted much from severe or protracted diseases (convalescence purpura); in the wasting, loss of elasticity, and vascular degeneration of the aged (senile purpura), and in the newly-born (purpura neonatorum).

Reviewing briefly the pathological conditions under which purpura occurs, we may arrange them as follows:—

I. (*a*) The infective diseases, in nearly all of which, but especially in small-pox, measles, scarlet fever, cerebro-spinal fever, syphilis, and malaria, purpura is an occasional incident.

(*b*) Rheumatism, which may be placed temporarily in this class, but requires separate description.

(*c*) The various conditions under which certain organic matters present in excess in the blood—such as bile, urinary constituents, or certain adventitious organic poisons, such as snake venom—may gain access to the blood.

(*d*) The presence in the blood of extraneous chemical substances, such as phosphorus, mercury, mineral acids, salicylic acid, iodide of potassium. For clinical purposes group (*d*) should be considered apart as "toxic" or "drug" purpura, but pathologically it fits in here.

(*e*) Conditions in which some constituent of the blood is wanting, as in scurvy.

(*f*) Alteration in the formed elements of the blood, as in anæmia and leucocythæmia.

To Series I. the term "Vascular purpura" may be given.

II. (*a*) Conditions that offer an impediment to the circulation, general or local; as in diseases of the heart and large vessels, and tumours compressing them, thrombosis, temporary vascular spasm or paralysis; as in convulsive seizures, whooping-cough, or angina pectoris.

(*b*) Want of mechanical support of blood-vessels, as in wasting, in the newly-born, and the aged.

Series II. may be designated "Mechanical purpura."

III. Conditions in which the direct influence of the nervous system can be traced, as in tabes, neuralgia, and the like. To this series the name "nervous purpura," or purpura of nervous origin, may be applied.

IV. Congenital imperfection of the blood-vessels, as in hæmophilia—"hæmophilic purpura."

In the absence of a common cause, of a definite clinical course, of constant pathological changes, it is obvious that purpura is not a consistent or uniform symptom group, but is itself a symptom entering not into one only but into many groups.

The best notion of the circumstances in which purpura occurs will be conveyed by an analysis of 200 cases from the records of the London Hospital. They were not selected, but taken consecutively, so far as the records permitted. They are given in the following table:—

TABLE of 200 Cases of Purpura in the London Hospital, arranged as regards probable Causes or associated Conditions.

	Males.	Females.	Total.
Rheumatism	33	28	61
Doubtful rheumatism	7	3	10
Bright's disease	7	2	9
Heart disease	3	5	8
Anæmia	3	3	6
Leucocythæmia	1	0	1
Scurvy	6	0	6
Privation and dietetic	3	2	5
Pyæmia	0	2	2
Ulcerative endocarditis	2	0	2
Malaria	0	1	1
Rickets	1	0	1
Whooping-cough	0	1	1
Congenital syphilis	1	1	2
Tuberculosis	3	1	4
Alcoholism	2	0	2
Toxic (drugs)	3	0	3
Cirrhosis of liver	1	0	1
Convalescence	2	0	2
Injuries	2	0	2
Hæmophilia	0	1	1
Varicose veins	0	1	1
Peripheral neuritis	1	0	1
Unexplained	31	37	68
Totals	112	88	200

This table does not present any instances of purpura in connection with the specific fevers, for these, with the exception of enteric fever, are not admitted. Nor are there any cases of P. neonatorum or P. senilis.

Age incidence will be best shown by the subjoined table:—

TABLE of 200 Cases of Purpura arranged in Decades.

	Up to 10 years.	11 to 20.	21 to 30.	31 to 40.	41 to 50.	51 to 60.	61 to 70.	Totals.
Males	24	29	31	13	9	2	4	112
Females	25	28	17	10	7	1	0	88
Totals	49	57	48	23	16	3	4	200

From these figures, which fairly represent the condition in which purpura occurs, apart from the eruptive fevers and in the newly-born, it will be observed that purpura is more common in the male than in the female sex, in the proportion of 14 males to 10 females—not quite 1½ males to 1 female. This holds good for all ages with the exception of the first decennium, in which the females exceed the males by one. It will also be observed that the greatest number of cases occur in the first three decades, 77 per cent in persons under 30 years of age. In the fourth decade the numbers rapidly fall to less than half of those in the third decade; still fewer cases occur in the fifth decade, and only 7 cases occur in persons of either sex over 50 years of age.

The number of cases here dealt with is probably larger than in any published series; but it will be seen how comparatively rare purpura is when I say that 200 cases represent the number occurring amongst 63,834 medical cases in 16½ years. They only amount to 0·3 per cent of the medical cases, and this is probably a fair calculation of its occurrence in purely medical practice.[1]

The great variety of supposed causes or associated conditions is sufficiently striking. Still more so is the fact that in one-third of the cases tabulated no explanation was afforded for the purpura, though in several of the cases a necropsy was made. It will thus be seen how extremely complex is the pathology of purpura. All we can do in the present state of our knowledge is to accumulate further information, and to exhaust every means—histological, bacteriological, and chemical—in the investigation of cases. It will be observed that in by far the majority of cases in which anything definite can be ascertained as to the causation of purpura this is of a vascular character—some known or probable alteration of the blood, or some condition which brings about a change in the blood-vessels; and, arguing from the known to the unknown, it seems probable that, in those in which no definite causation can be ascertained, purpura is due to one of these two kinds of change.

Symptoms. — Certain phenomena are common to most cases of purpura.

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[1] A very few cases, too few to affect the calculation, were omitted as the notes were incomplete.

Changes in the extravasated blood.—Recent extravasations appear of a more or less bright red or crimson colour. They are usually oval or round, but may occur in lines or streaks—vibices. In a short time they become of a dull purple, and later of a brownish red tint; lastly, a brownish stain persists for a considerable time. In some cases a bluish green colour is present. In quite superficial hæmorrhages, spots, as they fade, present a yellowish hue, passing into a faint brown. As regards the changes in the blood effused, when the hæmorrhage takes place into the cutis, there begins, according to Unna, very soon after the occurrence of the bleeding solution of the hæmoglobin, which is partly reabsorbed with the blood-plasma, and partly crystallised in the tissue (precipitated). Where large masses of blood corpuscles are closely packed, they break up, without previously giving up their hæmoglobin, into yellow or brownish flakes, which are gradually converted into pigment granules, and as such are partly taken up by the connective tissue cells.

Pyrexia.—A certain degree of fever is present in more than half the cases. In the majority it is slight and transient; in others the disease runs a moderately febrile or highly febrile course (P. febrilis), and hyperpyrexia has been known to occur. The decidedly febrile cases are nearly always characterised by a greater severity, and are therefore attended with greater danger than those which are non-febrile; otherwise no important differences are noticeable.

Albuminuria, apart, of course, from cases in which it is plainly secondary to Bright's disease, is of rather frequent occurrence in purpura; it occurs in both febrile and non-febrile cases, and corresponds with the statement (p. 568) that in fatal cases the kidneys are frequently found diseased.

Digestive system. — Derangement of the stomach and intestine is common. Apart from anorexia, which is frequent, colic in severe paroxysms, vomiting and diarrhœa are so pronounced in some cases as to have been constituted into a special form of the disease (Henoch's purpura).

Hæmorrhages. — Hæmorrhage may occur from any of the mucous surfaces. Epistaxis is the most common; next, hæmorrhage from the gums and throat, and, following these in frequency, from the intestines, urinary passages, stomach, lung, and sexual organs. Intra-visceral and interstitial hæmorrhages also occur; and hæmorrhages in the retina may be detected during life by the ophthalmoscope. Retinal hæmorrhages are, however, rare in purpura.

On account of the variety of circumstances in which purpura is met with, some classification in the investigation of cases is absolutely necessary. Dr. Radcliffe Crocker's classification has a pathological basis; he makes the following varieties: (i.) Certain blood alterations; (ii.) visceral disease; (iii.) want of support to the vessels; (iv.) sudden changes in the circulation; (v.) diseases of the nervous system.

Dr. Pringle makes the following classes: A. Symptomatic purpura: (i.) Mechanical, due to increased blood-pressure; (ii.) dependent on changes in the blood or walls of blood-vessels; (iii.) toxic; (iv.) due to disordered innervation; (v.) the result of a specific infective virus; (vi.) cases which cannot be considered as belonging to any of the foregoing classes, and which must provisionally be classified as idiopathic. B. Purpura simplex. C. Purpura hæmorrhagica.

Professor Osler gives a very good provisional grouping of the varieties of the condition. A. Symptomatic purpura: (i.) infective, (ii.) toxic, (iii.) cachectic, (iv.) neurotic, (v.) mechanical. B. Arthritic purpura: (i.) a mild form known as P. simplex, (ii.) peliosis rheumatica, (iii.) Henoch's purpura. C. Purpura hæmorrhagica.

The following kinds will be described here:—(i.) Purpura simplex, (ii.) purpura hæmorrhagica, (iii.) purpura rheumatica, (iv.) iodic purpura, (v.) Henoch's purpura.[1] There is no fundamental distinction between P. simplex and P. hæmorrhagica; the former is a mild form of purpura, the latter a severe purpura with hæmorrhages from mucous surfaces. Both are symptomatic of a great variety of causes.

PURPURA SIMPLEX.—With or without preceding constitutional disturbance, hæmorrhagic extravasations make their appearance in the skin. They frequently begin in the lower extremities, but become generally disseminated over the whole surface. They may present a rough symmetry, or have a random distribution. The spots are generally circular or rounded, but may occur in streaks; they vary in size from mere petechiæ to extravasations as large as half a crown or larger. The attack may be ushered in by a slight rise of temperature, or febrile disturbance may arise in the course of the attack. Many cases are non-febrile throughout their course. The disease is most common in young persons. The patient may be anæmic, or may present a healthy appearance, and be well nourished. There may be some malaise, digestive troubles, and other constitutional disturbance; or these may be wanting. The first spots fade, passing through the changes of colour described, and new ones appear; so that all varieties of colour are present. After lasting a variable and indefinite period, usually a week or two, no fresh spots make their appearance, the old ones fade, and the attack comes to an end, leaving pigmentation of the skin where the hæmorrhages have been present, for some weeks or longer.

PURPURA HÆMORRHAGICA (*Morbus maculosus Werlhofii*).—The etymologically meaningless name P. hæmorrhagica—for all purpura is hæmorrhagic—is applied to cases in which not only cutaneous extravasations are present, but in which hæmorrhages take place from mucous surfaces also. It represents the more severe and dangerous kind of purpura. No more than P. simplex is it to be regarded as a uniform symptom group, for it occurs under a variety of conditions.

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[1] Neurotic purpura, or purpura of nervous origin, cannot be made into a well-defined variety; but the name neurotic purpura may be applied to cases in which the hæmorrhages can be confidently attributed to nervous influence. Dr. Weir Mitchell has described cases of neuralgia in which hæmorrhages occurred in the skin about the penis; Strauss (16) and others, purpura in connection with tabes. Purpura is also met with in angina pectoris, meningitis, whooping-cough and epilepsy. In the latter categories the immediate mechanism is probably vascular, and consists in a local increase of blood-pressure.

It may begin with more or less constitutional disturbance—headache, debility, gastric pain, and vomiting, and be followed by extravasations into the skin and mucous membrane, and free hæmorrhages from the latter. Or it may begin as P. simplex and later become P. hæmorrhagica, as bleedings take place into and from the mucous surfaces. The hæmorrhages vary in size as in P. simplex, but tend to be larger, and are often accompanied by hæmorrhagic œdema in large patches—as large as the hand or larger—appearing in certain parts, raised, reddish, or purple-blue in colour, and pitting on pressure. The orbits, the penis, and scrotum occasionally become extremely swollen, and the skin tense and of a livid colour. The appearance may suggest a fear of sloughing, and indeed the fear may be justified. The cutaneous hæmorrhages pass through the same stages as in P. simplex, but appear in rapid succession, and are often of large extent. Hæmorrhages in severe cases are usually met with in the mucous membrane of the mouth and throat; and in this situation they may give rise to alarming symptoms, and even occasion a fatal issue. In several recorded cases hæmorrhages have taken place into the palate and tongue. When occurring in the latter organ acute swelling of the tongue, resembling acute glossitis, has been produced, necessitating incisions for the relief of the consequent dyspnœa. In one or two cases sloughing of the tongue, with shedding of its apex, has occurred.

Of the hæmorrhages that take place from the mucous membranes epistaxis is the most common; hæmorrhages from the mouth and throat are also very common: in some cases hæmorrhages occur from the stomach, intestines, lungs, and genito-urinary organs. The bleedings from the mucous membranes may be very severe and frequently repeated, and in some cases are uncontrollable. Though in some cases anæmia may not be present at the outset, it rapidly makes its appearance, which is not surprising when we consider the large amount of blood lost from the mucous surfaces and into the skin. Von Laache has recorded a case in which, eighteen days after the beginning of the disease, the red corpuscles numbered only 2,680,000 per c.mm., and the hæmoglobin was 0·067 per c.mg. In another, a woman twenty-one years of age, the corpuscular richness was 2,091,900. When the disease has lasted some time, and, as would be anticipated, when copious internal hæmorrhages have taken place, the blood richness, in numbers and colour, shows still greater reduction. Hayem has recorded a case in which the red corpuscles fell below 1,000,000; Quinquand a case with only 740,000 per c.mm.; and Hérard a case in which the corpuscular richness was 1,885,000, when first counted, but fell to 620,000 per c.mm. I have recorded a case (10) in a child eleven months old, in which the red corpuscles just before death numbered only 290,000 per c.mm., or 5·8 per cent, with one white to fifteen coloured corpuscles. In this case the great debasement of the blood was due to uncontrollable epistaxis, and the patient died quite exsanguine. The blood that exudes in such cases of extreme anæmia is only tinged with red, appearing as a thin serous exudation.

Fever is present in the majority of cases of P. hæmorrhagica. It may reach a high grade—104° F. or higher, and may be hyperpyrexial—105·5° or higher. Such cases with high fever, extensive extravasations, and copious and repeated hæmorrhages from the mucous membranes, may run a very rapid course, and end fatally in the course of a few days. Such cases have been described as P. fulminans. In severe cases hæmorrhages may take place into the brain, and may occasionally be seen during life in the retina. In P. hæmorrhagica pains are often present in the joints and limbs, even in cases in which there is no reason to believe the condition to be of rheumatic nature. Schebey Buch has drawn attention to effusion into the joints in non-rheumatic cases. Albuminuria, with or without blood, is often present in cases of P. hæmorrhagica. In fatal cases pulmonary œdema, often associated with hæmorrhage into the lung due to exhaustion, is commonly the determining cause of death.

In cases which pursue a favourable course, or which do not end fatally, the hæmorrhages into the skin and from the mucous membranes recur from time to time over a period of days, or, more usually, of weeks, in a fitful manner, and eventually cease; the patient being left extremely weak, anæmic, and often much wasted.

PURPURA RHEUMATICA (Schönlein's *Peliosis rheumatica*).—This kind has gradually gained increased recognition, though twenty or fifteen years ago it was scarcely ever diagnosed. Schönlein's description is worth reproducing, as differences of opinion have arisen as to the meaning of the name he used.

"The patients have either already suffered from rheumatism, or rheumatic symptoms accompany the attack: slight periodic throbbing pains in the joints (in the ankles and knees, rarely in the hand and shoulder-joints), which are œdematously swollen and tender on pressure. The characteristic spots of the disease in the majority of cases first appear on the extremities, especially on the lower extremities, and here only as high as the knee (rarely on the upper). The spots are small, of the size of a lentil to that of a millet seed, bright red, not raised above the skin, *disappearing under the pressure of the finger* [italics not in original]; they gradually become dirty brown or yellowish, the skin over them slightly desquamates with a branny scale. The eruption comes out in crops, often during several weeks. Ever so slight a change of temperature, as for example passing into a colder room, may occasion a fresh outbreak. The eruption usually appears with some fever, of a remittent type. Towards evening the symptoms are at their height, with a recession in the morning. There is frequently a deposit in the urine." It is clear from Schönlein's own words that he described an erythema papulatum, for he expressly notes the colour "disappearing under pressure." Further, in discussing the diagnosis, he gives the diagnostic criteria from Werlhof's disease (P. hæmorrhagica). The majority of writers, following Schönlein, regard purpura rheumatica as a purpuric erythema, though this is scarcely justified from his description that the colour disappeared on pressure. Some go farther and appear to regard all purpura as erythematous in nature. Though an allied process, I believe it better to keep the two conditions distinct, and in the following description of purpura rheumatica I shall restrict the name to a condition which is purpuric from the beginning, and in which the spots do not disappear on pressure at any stage. In other respects Schönlein's description of the eruption coming out in crops, and of the aggravation of disease in the evening, is singularly apt.

The disease occurs with about equal frequency in the two sexes, and is most common in the second, third, and fourth decennia (from eleven to forty); it is rare before ten years of age and after forty. In some cases the purpuric eruption makes its appearance whilst the patient is suffering from acute or subacute rheumatism. More commonly the arthritic symptoms arise coincidently with the purpuric eruption; in a few cases, in which arthritic symptoms are doubtfully present in the attack, or are entirely absent, an attack of arthritic rheumatism may appear at some subsequent period, thus revealing the rheumatic nature of the purpura; or, perhaps, to be more exact, thus demonstrating that the patient is a rheumatic subject. Apart from cases in which acute or subacute rheumatism ushers in the purpura, the very characteristic onset and course of the disease is as follows:—The patient has pain in the lower extremities, which may be of a dull aching character, but frequently and characteristically is a sense of tension—a "sensation of bursting" in the parts affected, as patients frequently describe it; often there is itching. When these symptoms are present (and patients who have had a previous attack know well their meaning), bright red spots, which do not disappear on pressure, are seen on the legs. In the majority of cases when they first make their appearance they are raised (P. papulosa). The eruption and its accompanying discomforts usually make their appearance in the later part of the day, afternoon or evening. The knee and ankle joints are usually painful and often swollen and tender, sometimes the skin over them is slightly reddened. A slight degree of œdema of the lower part of the leg, of the ankle, and of the dorsum of the foot is present in nearly all cases. By the following morning the pain remits, and inspection shows that the spots are now of a purple or dull red colour, and no longer raised. On the second evening, or after an interval of two or three days, the same phenomena are repeated—the aching of the legs, the pains in the joints and œdema, and the appearance of another crop of bright red spots similar to those first observed. The spots pass through the usual stages of discoloration characteristic of hæmorrhages into the skin, and if the patient is seen after the occurrence of two or three outbursts, and at a time when a fresh crop has recently appeared, we observe:—1. Bright red raised spots, varying in size from a millet seed to a threepenny piece or larger, not disappearing on pressure. 2. Spots of a similar size of dull red or purple colour, but not raised above the surface, and unaffected by pressure. 3. Yellowish brown stains. The affected limbs are tender to pressure and slightly œdematous. In most cases, as already stated, there are pain and swelling of the joints of the lower extremities, and in some of the elbows and wrists; even in cases in which the skin of these parts is not affected by hæmorrhages. The joint affection often persists between the outbursts of hæmorrhages, though exacerbations of pain and swelling occur in the attacks. The first outburst of hæmorrhages is usually confined to the lower part of the legs and feet. In subsequent outbursts there is a tendency to an extension of range, so as to involve the upper part of the legs, and, later still, the thighs and buttocks. In slight cases the eruption is limited to the lower extremities, but in more severe cases the forearms and arms are affected also. Usually when the thighs are affected the skin above and below the elbow is the seat of hæmorrhages. The eruption is so far symmetrical that if one leg is affected the other leg is affected also; and if it attacks the upper extremity both will be attacked. The eruption shows no marked predilection either for the flexor or extensor surfaces of the limbs. In the great majority of cases it is confined to the extremities; but in the more severe cases, especially those in which the arthritis and pyrexia are great, the trunk and face also are affected. Hæmorrhages into and from the mucous membranes are rare, but in a few cases small hæmorrhages may be seen in the buccal mucous membrane; and in rare and very severe cases extensive hæmorrhages may take place into the tongue (intra-muscular) and throat. In about one-third of the cases some degree of pyrexia is present, and probably, if careful thermometric observations were made in the evening at the time of the eruption, some elevation of temperature would be found in nearly all of them. Sweating is not a marked symptom unless the arthritis be pronounced. The amount of constitutional disturbance is variable, this being slight in the majority of cases; but in some malaise and debility are present. The majority of patients are able to walk about stiffly in the early part of the day, but locomotion is very difficult and painful in the later day, especially at the time of the outbreaks of hæmorrhages. Any exertion tends to bring on an attack. The department for diseases of the skin, of which I have charge, is open in the morning, and patients tell me they had a bad attack in the afternoon or evening of the days when they had been to see me. Exertion seems to me to determine attacks much more than changes of temperature, to which Schönlein referred them. The eruption usually lasts an indefinite time, unless treatment of a certain kind is adopted for several weeks, or months; I have known it to persist for two years. The disease is occasionally, though extremely rarely, fatal. Usually it is a benign affection. It is very apt to recur.

The assemblage of symptoms is very definite and characteristic. The occurrence of hæmorrhages—usually confined to the extremities, appearing in crops, usually in the latter part of the day—the arthritic pain and swellings, and its protracted course, distinguish it from other forms of purpura, and from erythema exudativum multiforme. It has undoubtedly close clinical alliances with the latter, which also, in a large proportion of cases, is of a rheumatic nature, and the two may occur in the same subject; but in the cases to which I would restrict the name purpura rheumatica the eruption from first to last is purpuric, and not erythematous. The evidence of its connection with rheumatism is, in the majority of cases, extremely distinct. The arthritis, which is present in many cases, is characteristic, and may precede the purpura; so that the diagnosis of acute or subacute rheumatism is already made. In other cases the patient has previously suffered from rheumatic fever. In a considerable proportion of cases valvular disease, usually mitral incompetence, is present; and in a few it may arise during an attack. Many patients have had other affections belonging to the rheumatic series, such as tonsillitis, endo- and pericarditis, pleurisy, chorea; and a family history of rheumatism is very common. I have seen two brothers with purpura rheumatica at some years' interval. The second one died of heart disease a few years later. In a certain number of cases the arthritic symptoms in the attack are equivocal or absent; but the complex of symptoms described have been definite and identical with those in which the rheumatic nature was beyond dispute; so that when the symptoms above described are present, I am of opinion that we are justified, even in the absence of arthritis, in diagnosing purpura rheumatica. I have seen a case in which no arthritis accompanied the purpura, yet (at an interval of a year or more) an attack of rheumatic fever subsequently appeared. In the list I have given of the ascertained causes of purpura, rheumatism, it will be observed, stands very high, giving in the 200 cases 30·5 per cent, exclusive of doubtful but still probably rheumatic cases.

IODIC PURPURA.—Fournier was the first to give a good description of this form of purpura. The eruption is generally confined to the lower extremities, and in the majority of cases to the parts below the knee. The eruption consists of discrete miliary hæmorrhagic spots, bright red when recent, not elevated, not obliterated by pressure, unattended with heat, pain, or swelling. The eruption comes out at an early period of the iodide treatment, and continues to appear for two or three days. It remains for a certain time as a staining of the skin, the blood undergoing the changes of colour usual in cutaneous hæmorrhages, and finally disappears by the end of two or three weeks. During its progress a renewed attack may sometimes be induced by augmenting the doses, and then the bright red recent hæmorrhages contrast very markedly with those that are fading. Though usually confined to the legs, it may affect the trunk and the face, as in a case I have recorded. The purpuric spots are usually more pronounced in the anterior than in the posterior parts of the legs. Successive outbreaks are usually less profuse than the original one. It may be accompanied by some œdema of the legs, but this is not usually the case. The various salts of iodine seem all to produce purpura, but exceptions to this rule are met with;

some persons can take sodium or ammonium iodide without inducing it, whilst potassium iodide is operative; or potassium iodide may fail to produce it, whilst ammonium iodide may be operative. As potassium iodide is the salt most frequently prescribed, purpura is most frequently met with in patients taking this preparation. It is held by Besnier that pure iodine will not cause the hæmorrhages, and he has illustrated this fact in the person of a man who had purpura in the lower limbs every time he took iodide of potassium; yet, although tincture of iodine caused symptoms of iodism in him, no purpura appeared (12). Purpura is a very rare consequence of the administration of potassium iodide. Usually it is quite a benign and unimportant affection, but one to be borne in mind, lest it be misinterpreted. Occasionally, moreover, the salt may give rise to very grave symptoms and even prove fatal; as in a case I have recorded. In this case fatal purpura followed a single dose of 2½ grains of potassium iodide in an infant five months of age. In prescribing iodides to young children a small dose should be first given, and if tolerated, the dose may be augmented.

The reaction is clearly due to idiosyncrasy, as it occurs in a very small minority of persons. It does not depend on the debilitated state of the patient, whose nutrition may be quite good. It is possible that syphilis favours its occurrence; but the frequency with which iodides are administered for syphilis and the rarity of iodic purpura show that personal peculiarity or idiosyncrasy is the determining factor. It is probable that the cause of the iodide purpura is some chemical action of the drug on vaso-motor centres producing variations in pressure in the area in which purpura appears; but it is possible that the drug may have a selective action on particular vascular areas, rendered more vulnerable than usual by incidental influences.

HENOCH'S PURPURA.—Though Willan (21) many years previously had described a case of this kind very graphically, it was not until Henoch published a series of cases that attention was prominently directed to this form, often called "Henoch's purpura" (6). Couty recorded a number of cases which he recognised as similar in nature to those described by Henoch; and Osler, who takes a more general view of purpura than I have done in the present article (including it under erythema exudativum), has particularly directed attention to the visceral complications.

The marked feature of this disease is the association of abdominal symptoms (vomiting, colic, intestinal hæmorrhage) with purpura and arthritic swellings. The attack may begin with rheumatic pains and swellings of the joints, and be followed by purpura and colic with vomiting and blood in the stools. Or it may begin with gastro-intestinal derangement, and the purpura and articular swellings and pains follow. What is especially characteristic of it is the occurrence of repeated outbreaks of colic, vomiting, and hæmorrhage from the bowels, with purpura and pains and swellings in the joints. The illness generally consists of a series of such events over a period of some weeks or months; but intervals of months may occur, and fresh outbreaks then take place. Recurrence is one of its most characteristic features.

The colic is generally of a very intense character. The abdomen is usually tender, especially over the colon. The vomiting is often severe and protracted, frequently bilious, occasionally but not frequently bloody. The stools contain more or less coagulated blood, but in some of the attacks no blood may be passed. In some cases albuminuria is present, and well-marked symptoms of nephritis set in which may prove fatal; or this complication may slowly subside. Epistaxis, hæmaturia, hæmoptysis may occur, but are not frequent. In the majority of cases the eruption is purely hæmorrhagic, but in others, in addition to the purpura, purpuric œdema, exudative erythema and urticaria may be present. Most commonly the eruption is confined to the extremities, but it may involve the face and trunk; and hæmorrhages may occur in the mouth and throat. In the attacks the joints are usually affected. There may be only pain and stiffness, or there may be effusion and redness of skin over the articulations. The dorsa of the feet are often swollen, as in purpura rheumatica. There is as a rule little pyrexia, and it may be entirely absent. In one of Osler's cases great coldness of the feet was a prodromal symptom of the attacks, and in one case the spleen was enlarged. Silbermann has published a fatal case (7): a child, aged ten years, was attacked on December 15, 1887, with fever and pains in the knees. On the 16th there was an outbreak of purpura, with colic, hæmatemesis, and melæna, and after persisting for three days the symptoms disappeared. The attack recurred in January with great severity, and on the 20th, 21st, and 22nd there were signs of peritonitis. The autopsy showed an acute peritonitis, which had resulted from a perforation at the fundus of the stomach. There was no ulceration in the bowels, but the mucosa was swollen and congested. There were necrotic foci in the stomach and intestines, and thrombi were found in some of the blood-vessels.

Henoch's purpura is relatively most common in childhood, but it occurs in adults also. As to the nature of such cases the evidence is inconclusive, and whether the colic and vomiting stand in relation to the hæmorrhage from the bowels and stomach as cause or effect is uncertain. Silbermann's case, however, suggests that hæmorrhage is the primary event and may lead to ulceration and perforation; as hæmorrhage is an exceedingly rare or almost unknown event in colic of the most severe degree, as in lead poisoning. Some of the cases appear to be of a rheumatic nature—the patients, as in some recorded by Henoch, having previously had a rheumatic attack without purpura or colic. Couty regards the disease, by the exclusion of other causes, as of nervous origin, affecting the vaso-motor nerves.

Diagnosis of Purpura.—It must be reiterated that purpura is rather a symptom than a disease. It is not sufficient, therefore, to recognise purpura, but the nature of the process in the individual case must be ascertained. To recognise the symptom purpura is an extremely easy matter. The occurrence of hæmorrhages in the skin and mucous membrane is demonstrated by an eruption of blood-colouring matter not of traumatic origin, the colour not disappearing under pressure. In many forms of exudative erythema there is blood extravasation, but this is accompanied by overfilling of the blood-vessels, which may be emptied by pressure, the colour returning when the pressure is removed. To this condition the name purpuric erythema may be applied; but the name purpura should strictly be applied to cases in which the hæmorrhages are primary, unattended with erythema, and not due to injuries.

Having decided that purpura is present the observer has next to search for its cause. It is for this reason that some clinical classification is not merely desirable, but absolutely essential. The first step is to ascertain whether the purpura is an expression of one of the specific diseases prone to be attended with hæmorrhage. Small-pox, scarlet fever, measles, pyæmia, syphilis, and rheumatism have especially to be borne in mind. The diagnosis of purpura rheumatica has been sufficiently given. Next, the various primary blood diseases have to be considered—pernicious anæmia and leucocythæmia in particular. The appearance of the patient may afford a clue, but the most important matter is the examination of the blood. In the next place the possibility of scurvy must be remembered. In this disease, in addition to the cutaneous hæmorrhages, subcutaneous and intra-muscular extravasations occur, producing brawny, blood-stained patches in the hamstring muscles and calves of the legs, and in the skin over the patches; and the gums are swollen and bleeding. Evidence of insufficiency of fresh vegetable or animal food is generally obtainable. In doubtful cases the effect of treatment will assist in the diagnosis, as scorbutic cases rapidly improve when treated with fresh vegetables and meat juice. It must be remarked, however, in this connection that in certain cases of pernicious anæmia the gums may be swollen and bleeding as in scurvy. To these cases the name "scorbutic anæmia" has been applied. Scurvy being excluded, the possibility of some poison having been accidentally or intentionally taken must be considered—phosphorus, mercury, mineral acids, etc., being borne in mind; or some medicinal substance, especially iodide of potassium. Nor must the possibility of a nervous origin be forgotten; the history of the case, and an examination of the nervous system, will determine whether this cause be in operation.

Finally, in a considerable number of cases no definite cause can be ascertained for the purpura, and such cases are indicated by the name P. idiopathica. They must temporarily be relegated to the class purpura simplex or purpura hæmorrhagica, according to the symptoms presented. It must be remembered, however, that this indefinite name serves but to remind us of our ignorance; and the observer must ever be on the alert to discover the cause which will immediately remove the case to its own category.

Prognosis.—Most cases of purpura end in recovery. The mortality is probably about 14 or 15 per cent. Thus of the 200 cases above analysed the mortality was 28, or 14 per cent. Sex does not appear to exercise any decided influence—the mortality in males, in the 200 cases, being 14·2, whilst in females it was 13·6. Age exercises some influence, the gravity appearing to increase, on the whole, with the age of the patient. But in the 200 cases analysed the mortality in the first decade was 16 per cent; in the second decade, 10 per cent; in the third decade, 14 per cent; in the fourth decade, 13 per cent; in the fifth decade, 25 per cent; in the seventh decade, 25 per cent. It is thus seen that the greatest mortality occurs in patients under 10 and over 40 years of age. Cases of P. simplex almost invariably end in recovery; but, on the other hand, purpura simplex may be the beginning of a severe and fatal case of purpura hæmorrhagica. The gravity of the case is generally stamped early upon it. The severity and frequency of the cutaneous hæmorrhages, the occurrence of hæmorrhages from mucous surfaces, the degree of pyrexia, the constitutional depression, the degradation of the blood, as proved by the hæmoglobinometer and hæmocytometer, and the occurrence of marked albuminuria, will draw attention to the danger attaching to the case. At the same time it must be borne in mind that the most severe. and apparently dangerous cases sometimes end in recovery.

Treatment.—The patient in all kinds of purpura should be confined to bed. Whenever a definite cause for the purpura is discoverable, a clue to the treatment will be supplied. In the infective diseases it invariably indicates a very grave condition, and calls for support by nourishing fluid food and stimulants; whilst at the same time some of the hæmostatics to be subsequently mentioned, especially turpentine, should be administered. In syphilitic purpura iodide of potassium should not be given, for it appears in some cases to increase or to initiate the hæmorrhages. In many cases of P. rheumatica oil of turpentine appears to act as a specific; it should be given in ten or twenty minim doses in capsules, or suspended by means of tr. quillaiæ saponariæ or mucilage. The following mixture I have used in numbers of cases with the happiest results:—Ol. terebinth ♏ x., quillaiæ sapon. ♏ x., aquam cassiæ ad ℥j. To be given three times a day. Prof. Boeck of Christiania recommends antifebrin in five or ten grain doses in these cases. Salicylates, in my experience, as well as in that of Prof. Boeck, have little or no influence for good.

In cases in which no discoverable cause can be ascertained, as well as in many in which there is a recognised cause, turpentine is, on the whole, the best remedy. Ergot and hamamelis may be tried, but they have not proved very efficacious in my hands. Calcium chloride, suggested by Dr. Wright of Netley, is indicated when deficient coagulability of the blood is proved or suspected, and certainly should be tried when other remedies fail. It should be given at first in twenty-grain doses, every three or four hours, the dose being reduced later to fifteen or ten grains; as when given in excess it diminishes the coagulability of the blood. Iron, as preparations of the perchloride or persulphate, appears useful in some cases in the attack, and should be given in convalescence to remove the anæmia which so commonly results in severe cases. In Henoch's purpura Henoch himself has seen benefit from an ice-bag applied to the abdomen;

in chronic cases he recommends perchloride of iron. Osler in two cases saw benefit from arsenic, which appeared to control the tendency to recurrences. In other cases, however, it failed.

Petechiæ.—Minute hæmorrhages in the skin, the size of a flea-bite. Such hæmorrhages vary in colour from bright red, dark red, to purple; and have this characteristic, that the colour does not disappear on pressure. Petechiæ may be one of the expressions of purpura, in which case they will be almost invariably associated with larger hæmorrhages in the skin, and in these circumstances own the most varied causes (see Purpura). Or they may be caused by the bite of the common flea. In the latter case, when recent there is a small circular spot of erythema with a pin-point hæmorrhage in its centre. The erythema disappears on pressure, to return when the pressure is removed, whilst the central punctum remains, as it does also when the erythema spontaneously subsides. When due to flea-bites, recent lesions, with the above characters, will almost invariably be found affording a clue to their nature. Their position, on covered parts of the skin, as well as the evidence of want of personal cleanliness of the patient and clothes, will be of assistance in determining their nature. There is some evidence that cachectic conditions and want of food favour the persistence of the minute hæmorrhages due to flea-bites, and their abundance undoubtedly implies neglect. When associated with pyrexia they may cause mistakes in diagnosis, especially as regards typhus and measles; so that the subject is not unimportant. Care with regard to the above points will enable the observer to avoid errors in diagnosis. The term petechial is applied to diseases, especially fevers, accompanied by hæmorrhages.

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SCURVY

SYNONYMS.—Lat. *Scorbutus;* Fr. and Germ. *Scorbut;* It. *Scorbuto;* Russ. *Zinga.*

SCURVY is a general apyretic and non-contagious disorder consisting of mental depression, extreme debility, a tendency to syncope, and special lesions of the mouth, skin, and muscular system, indicative of a morbid change in the composition and properties of the blood. Of these lesions the most frequent and most marked are swollen deeply congested and softened gums, petechiæ and diffused livid patches on the surface of the skin, and swelling and rigidity in the hams. In severe and advanced cases there may be bleeding from the mouth and nose and from internal organs, and rapid breaking down of ulcerated, injured, or scarred skin.

Scurvy is still endemic in certain small districts in the north-east of Europe and in Asiatic Russia. It has occurred from time to time on land in epidemics, differing in extent and severity in different instances, but invariably produced under analogous conditions. The disease seems to have preserved the same type, and the records of recent outbreaks show that it is capable now of presenting characters equal in virulence and intensity to those recorded in past ages. The history of land epidemics proves clearly that it is very seldom met with save in times of war and famine, or under circumstances of neglect; and that it should always be dreaded in besieged towns, in armies in the field, after a widespread failure of crops, and in badly-provisioned and overcrowded public institutions. These conditions being present, scurvy will not spare the members of the most advanced and civilised communities. Paris suffered severely during the last siege, and both the French and English armies were much stricken in the Crimean War. Of about 110 records of epidemics of scurvy in the course of the present century, collected by Hirsch, 11 occurred in Great Britain.

It is chiefly from its former prevalence at sea that scurvy has excited the most interest. To the recorded experience of naval medical officers the profession is indebted for most of its knowledge of the nature of the disease, and, from their successful efforts to banish this grievous scourge from the service, it has learnt not only how to treat, but also how to prevent it. The oft-quoted passages from the history of Lord Anson's expedition in 1740 gave but a partial idea of the ravages caused by scurvy in the Royal Navy in the course of the past century. According to Lind, it killed more men than did the hostile French and Spanish armies; and in 1795 the safety of Lord Howe's fleet was seriously endangered by an outbreak of this disease. From this date, when, at the recommendation of Sir Gilbert Blane, lime-juice was introduced into the Navy, scurvy has gradually decreased; and during the past fifty years, except in some few outbreaks arising under exceptional circumstances, it has become so rare as to be practically abolished as an important disease (Bryson).

Notwithstanding this example and the striking results from the adoption of so simple a preventive measure, scurvy, until quite recently, prevailed to a very unsatisfactory extent in merchant ships. In 1864 it was pointed out by Dr. Barnes that, during the twelve years following 1851, 1058 cases of scurvy had been admitted into the hospital ship *Dreadnought*. The following table, giving the numbers of cases subsequently admitted into this institution, shows a gradual but interrupted decline, which, during the past six years, has reached such a point as almost to justify the hope that this disease will soon be practically extinguished in the British merchant service as well as in the Royal Navy:—

TABLE of Cases of Scurvy treated in the Seamen's Hospital, Greenwich, from 1864 to 1896.

Year.	Cases of Scurvy.	Fatal Cases of Scurvy.	Year.	Cases of Scurvy.	Fatal Cases of Scurvy.
1864	74		1881	36	
1865	101	2	1882	28	
1866	96	5	1883	15	
1867	90	3	1884	6	
1868	64		1885	8	
1869	31		1886	5	
1870	30		1887	12	1
1871	21		1888	10	
1872	30		1889	2	
1873	7		1890	3	
1874	18	1	1891	4	
1875	15		1892	0	
1876	30	1	1893	3	
1877	24	1	1894	1	
1878	30		1895	0	
1879	21		1896	3	
1880	46				

Of the 302 cases admitted in the course of the past twenty-two years, 240 were brought from British and 62 from Colonial and foreign vessels.

Scurvy may occur in any climate; and neither extreme heat, nor extreme cold, nor excess or absence of humidity, is to be regarded as an essential factor in the causation of the disease. Though more frequently observed in northern and cold regions it has at times prevailed severely in India and China, and amongst exploring parties in Australia. It attacks in the same way both white and coloured subjects, and no race is exempt. Its greater prevalence amongst adult males is doubtless due to incidental circumstances, as in extensive epidemics on land neither sex nor age affords immunity or even resistance against attack.

Etiology.—There can be no doubt that this disease, though almost invariably associated with circumstances of privation, is the result of a defective quality of food, and not merely of a reduced supply. The large majority of those who have had actual experience of scurvy, and have carefully studied the records of its epidemics, are convinced that the defect consists mainly in the want of vegetable matter, which forms part of every ordinary and adequate dietary. Whether, as it is held by some, scorbutic symptoms may under certain circumstances be due to the absence of fresh animal food is still an unsettled question, and so must necessarily remain until more is known of the essential nature of the disease. Notwithstanding the doubts of Immermann and Mahé, and the expression of opinion by the medical members of the Arctic Survey Committee, in 1877, that scorbutic disease may be due to an absence of fresh meat, it seems difficult, on a review of the evidence that has accumulated since the middle of the last century, to resist the conclusion, first formulated by Bachstrom, that the primary and only cause of this disease is an absence of vegetable food. The question is one of purely scientific interest, and need not at present be brought to bear on measures of prevention and treatment. Whatever may be the differences of opinion as to the causes of this or that epidemic, there is an absolutely unanimous agreement, amongst both medical men and ship's officers, that the only sure and effectual means of preventing this disease, and of curing it when it has shown itself, is the supply of fresh succulent vegetables or fruits, or of a pure vegetable juice. As the introduction of lime-juice into the Royal Navy in 1795 was speedily followed by a practical extinction of scurvy, so in recent years a like result has been attained in the merchant service by securing for seamen a good supply of this antiscorbutic, and by a general adoption of a dietary of increased vegetable and reduced animal food.

If further evidence, beyond that collected and reviewed by Dr. Buzzard in 1870, were needed in support of the conclusion that scurvy is exclusively caused by the absence of vegetable nutriment, it would be found in the accurate and carefully prepared records of subsequent outbreaks. The appearance of scurvy in Paris in the winter of 1870-71 was due, as Delpech and Bucquoy showed, to a failure of vegetable and not of animal food; and in the thorough inquiry into the causes of scurvy in the Arctic Expedition of 1875-76, the outbreak was unanimously attributed by the members of the Admiralty Committee to the absence of lime-juice from the sledge dietaries. The latest official returns of scurvy on board British merchant ships also support the same conclusion.

It is necessary to bear in mind that the manifestation of scurvy, as of other constitutional disorders, especially those of a cachectic character, may be much favoured, though not directly caused, by such conditions as are likely to impair physical vigour, and to disturb the maintenance of good health. Amongst the host of such indirect and remoter causes mention may be made of exhaustion by hard work, poor diet, previous disease, faulty hygienic conditions such as bad air and water and overcrowding, deprivation of sunlight, monotonous diet, and an almost exclusive use of salt meat. In instances of scurvy on board ship, debility from previous disease, especially dysentery and malarial fever the most frequent penalties of tropical service, often plays an important part; and very frequently the first manifestations of a scorbutic taint are excited by extreme cold, or by a sudden transition from warm to cold and rough weather. The latter conditions probably exert in most cases a mixed or indirect influence, as cold and bad weather at sea usually necessitate increased work and exhausting or prolonged muscular exertion.

On the other hand, such conditions as a more or less varied diet, freedom from severe physical labour, a good standard of health and vigour, and fair hygienic surroundings will enable those who have been long deprived of vegetable food to resist and even to escape an attack of scurvy. Although it is not strictly true that this disease is exclusively one of the forecastle, there can be no doubt that even in the most severe outbreaks of scurvy at sea the number of officers affected is relatively very much less than the number of men. The existence of absolute immunity from scurvy under a very long-continued or habitual deprivation of vegetables has yet to be proved. It is not impossible, however, that in individual instances immunity may exist from scurvy as from acute infectious disease and many forms of organic poisoning. This quality of immunity, on which so much stress has been laid by some, does not affect the validity of the generally recognised rule as to the causation of scurvy; for, as was pointed out by the Arctic Commission, "although a deficiency or entire absence of fresh vegetable food is an invariable antecedent of a scorbutic attack, it does not follow that the disease invariably occurs during this deficiency or absence."

General pathology.—Inquiries into the general pathology of scurvy have hitherto consisted in attempts to determine on the one hand the changes in the blood and urine of scorbutic patients, and, on the other hand, the special chemical element of food the absence of which results in the appearance of scorbutic symptoms. Experimental researches on animals, as might have been anticipated, have failed to throw any light on this subject. The experiments of Stricker and Prussak on frogs seem to have been made in ignorance of the physiological peculiarities of these animals; and those of Hoffmann, quoted by Ralfe, have but an indirect bearing on the question.

No satisfactory information has yet been attained by the examination of blood and urine. The statements, as a rule, are very contradictory, the results of one observer being diametrically opposed by those of another. This is doubtless due to the difficulty of obtaining a sufficient quantity of either of these fluids for the purpose of strict scientific investigation. At the present day it would be considered unjustifiable to treat a scorbutic subject by bleeding; and the composition of the urine is always rapidly changed by the dietetic and remedial measures which, in a case of well-marked scurvy, it is necessary to prescribe without delay.

The next questions to be asked are what elements of nutrition are withheld in the absence of vegetable food, and to which of these, whether singly or in combination, the normal immunity from scurvy is to be attributed. We have been taught by wide experience that the most efficient of so-called antiscorbutics are fresh green vegetables, succulent and acescent fruits, and the juices of the latter, especially of the lemon and lime. Of vegetables in common use the most trustworthy are those represented by the lettuce, cabbage, potato, yam, onion, cress and other cruciferous plants. The most prized and useful fruits are such as are juicy, particularly those belonging to the order of Aurantiaceæ. Apples also are good antiscorbutics, and their use has done much to protect American seamen against scurvy. Vegetables retain their antiscorbutic properties when preserved, though to a diminished extent. Probably of all forms of preserved vegetable sauerkraut is the best. As substitutes for succulent fruits lime and lemon juice appear to be by far the most efficient. Malt liquors, spruce beer, and claret possess antiscorbutic properties, and probably cider also, which has certainly had a good reputation in this respect.

It seems to be quite clear that vegetables do not owe their antiscorbutic properties to their free organic acids. Citric and tartaric acids have been found practically worthless as antiscorbutics; indeed the use of the former as a substitute for lime-juice on board British ships has been legally proscribed. Though these acids are present in large proportions in the most succulent fruits, they exist but in small quantities in many antiscorbutic vegetables; and the potato, so it is said, contains no vegetable acids at all.

The simplest hypothesis of the causation of scurvy—which hypothesis, however, necessarily admits the antiscorbutic action of animal as well as vegetable food—is that based by Sir Alfred Garrod on the following conclusions which were published in 1848:—

1. That in all scorbutic diets (salt meat, leguminous vegetables, rice, bread, etc.) potash exists in much smaller quantities than in those which are capable of maintaining health.

2. That all substances proved to act as antiscorbutics contain a large amount of potash.

It seems strange that the well-known table on which these conclusions were founded has not been extended by further analyses of other articles of diet, especially of fresh vegetables and fruits. As it stands at present, the support it was to give to the hypothesis that scurvy is caused by a deficiency of potash in the food is much shaken by the fact that its data are opposed to those of common and repeated experience. Potatoes and lime and lemon juices are certainly excellent antiscorbutics, and contain large quantities of potash; but it is no less true that for the prevention and treatment of scurvy a vegetable diet, even though it may not contain these articles, is, to say the least, far superior to animal food; and that onions, for instance, possess far more active and useful antiscorbutic properties than salted or even fresh meat. Another and probably more serious objection is that, except in the case of potatoes, the arrangement of the few vegetables given in the table bears no relation at all to their comparative value as antiscorbutics. The administration of nitrate of potash, regarded by Dr. Buzzard as a crucial test, has failed both to prevent and to cure scurvy.

Immermann, who, following the late Professor Hirsch and other continental writers on scurvy, favours the potash hypothesis, tries to overcome these difficulties by suggesting that the scorbutic disorder may be due, not to an insufficient supply of potash to the organism, but to an absence or deficiency of this base in the tissues. An insufficient supply in the food, according to this author, is certainly one way, but not the only possible way, in which this absence of potash in the tissues can be brought about. In the first place, the potash combination may be supplied to the blood by the food in sufficient quantity, but in a form ill adapted for assimilation. Green vegetables and potatoes contain potash in easily assimilable form; whilst meat, leguminous vegetables, and bread contain the same alkali in a form less capable of decomposition and assimilation within the body. In the second place, a deficiency of potash in the tissues may arise in spite of an abundant supply in the food, when the food is prevented by intestinal disturbances, such as dysentery and diarrhœa, from entering the circulating fluid in sufficient quantities. In the third place, the absorption of the circulating potash by the tissue elements must also be influenced by those weakening agencies, such as deprivation of fresh air and light, want of exercise, excessive heat, and the like, which lessen the trophic energy of the cells, and diminish their capacity for appropriating the potash from the blood. The first of these explanations seems to be a very suggestive one, and likely, if it can be made good by further chemical investigation, to remove some of the most serious objections to Garrod's hypothesis. The second and third are opposed by the well-known clinical fact that in ordinary cases of scurvy a supply of fresh vegetables will speedily remove the purely scorbutic symptoms notwithstanding the persistence of associated diseases and other unfavourable conditions.

The view now in most favour with English authors, and one which seems capable of accounting for these numerous discrepancies, was expressed in a suggestion, made many years ago by Dr. Buzzard, that the antiscorbutic element in vegetable food is not potash alone nor the organic acids alone, but a combination of the two. Thus scurvy is to be attributed to the absence of organic salts of citric, tartaric, malic, and, as seems probable from an interesting Arctic record by Dr. W. H. Taylor, of oxalic acid also; especially of the potash salts, which are present in the growing leaves of plants and in fruits and their juices, and which in the organism are converted into carbonates. This view, as further explained and elaborated by Chalvet, whilst recognising the important part played by potash in the production of scurvy, solves many of the difficulties of Garrod's hypothesis. Potash in combination with citric, tartaric, malic, and (very probably) oxalic acids is readily absorbed, because the organic salts thus formed, being unstable, are converted into carbonates which are taken up into the organism, the potash being absorbed by the tissues, and the gas eliminated. In fresh meat and dry leguminous vegetables, on the other hand, the potash, though abundant, is much less useful for purposes of nutrition, as it is present in more stable forms such as those of chloride and phosphate. Thus, he says, mutton containing a given weight of potash fails to protect against scurvy, whilst lemon juice, containing not more of this base, acts as a most efficient antiscorbutic.

That this, however, is not the final and only possible view of the matter was shown by the late Dr. Ralfe, who, in an able paper on the general pathology of scurvy, endeavoured to extend the suggestions of Dr. Buzzard yet further. Ralfe, from observations on the results attending abstinence from fresh succulent vegetables and fruits, and from the analyses of urine from scorbutic patients, came to the following conclusions:—"The primary alterations in scurvy seem to depend on a general alteration between the various acids, inorganic as well as organic, and the bases found in the blood, by which (*a*) the neutral salts, such as the chlorides, are either increased relatively at the expense of the alkaline salts, or (*b*) that these alkaline salts are absolutely decreased. This condition produces diminution of the normal alkalinity of the blood; and it is suggested that this diminution produces the same results in scurvy patients as happens in animals when attempts are made to reduce the alkalinity of the body (either by injecting acids into the blood or feeding with acid salts); namely, dissolution of the blood corpuscles, ecchymoses, and blood-stains on mucous surfaces, and fatty degeneration of the muscles of the heart, the muscles generally, and the secreting cells of the liver and kidney."

From the results of his investigations Ralfe was led to surmises rather than to positive conclusions concerning the changes in the blood and urine of scorbutic patients. These surmises, however, are very suggestive, and likely to prove of much value as indicating the lines on which further researches of this kind should be carried out.

Symptoms.—Scurvy, as a rule, comes on slowly and insidiously; and the appearance of its external lesions is usually preceded by a preliminary stage of extreme physical weakness and mental apathy. In this stage the nature of the illness is indicated by shortness of breath, fleeting pains in the back and lower limbs, and a peculiar sallowness of the skin. As the morbid condition is displayed and the characteristic signs of scurvy manifest themselves, the following symptoms appear:—The patient is listless and weary; the skin is dry and rough, and marked by small purple spots (petechiæ), which are most abundant on the thighs and legs and, in many instances, are met with on the lower limbs exclusively. In addition to these spots there are livid patches of varying size and shape, which resemble bruises. Here and there, most frequently in the soft parts of the ham and calf, and behind the ankle, firm subcutaneous swellings may be felt which are widely diffused, are not well defined at their margins, and are very tender. The eyelids are slightly swollen, and the conjunctivæ often marked by bright red patches of ecchymosis. In some cases the eye is covered by the swollen and purple lids, and the conjunctiva presents the appearance described by

Dr. Buzzard as "tumid and of a brilliant red colour throughout." The lips are pale and anæmic, and the gums of a deep red colour, very soft and vascular, and much swollen. The tongue is moist and clean. There is a peculiar and characteristic fœtor in the breath. The patient suffers from breathlessness, which is increased by the slightest muscular exertion; he sleeps well and retains a fair appetite. The urine is scanty, and the bowels are usually constipated.

Of these characteristic symptoms of a mild and ordinary scorbutic attack, the earliest and most frequent are those presented on the surface of the skin. In the primary stage, and when all other signs of scurvy are absent, there will be found almost constantly a dirty and pale yellow stained skin, and a decided dryness of the epidermis with a tendency to desquamation. Duchek has directed attention to the frequent presence of a roughness over the extensor surfaces of the limbs caused by elevation of the follicles. The petechial spots, each of which is formed by a small and circumscribed effusion of blood around a hair follicle, are smooth, level with the surface of the skin, and persistent under digital pressure. The centre of each is usually traversed by a hair. These spots are in most cases confined to the lower limbs; but in a severe and prolonged attack they may arise on other parts of the body, with the exception of the face. The patches of ecchymosis which usually appear later than the petechial spots, and are not constant, are likewise met with most frequently in the lower limbs: although within these limits they have no special seats of election, they often occur just over or near a large subcutaneous swelling. Like the patches of ecchymosis observed in the subjects of hæmophilia, they are probably due to slight injuries. The tender subcutaneous swellings which occur so frequently in the popliteal space and the muscles of the calf, and which are sometimes met with in the sheath of the rectus abdominis muscle and the armpit, usually succeed the more superficial lesions.

The affection of the gums and the subcutaneous indurated swelling are the two especial lesions of scurvy. The former, though generally regarded as a test symptom, is by no means constant. In most cases it is an early and well-marked symptom; but sometimes, even though all the other lesions may be present in a severe and advanced form, this may be altogether absent, and the gums may remain smooth and regular, though very anæmic, and of a pale blue colour. The swelling and discoloration may come on suddenly and increase rapidly at an early stage of a scorbutic attack, or may advance very slowly whilst all other symptoms are well marked. The intensity of the gum affection, though, as a rule, most marked in very severe and advanced cases of scurvy, often fails to bear any proportion either to that of the general condition or of the other local symptoms. The first indications of the gum affection are usually redness and swelling of the tongue-shaped extensions of gingival tissue between the teeth. Afterwards the gums along the dental arches, both in front and behind, form soft and pulpy swellings of a deep red colour, which are tender and bleed readily when rubbed. Where any teeth are absent, there is little or no swelling; and in very old or young subjects who are edentulous the morbid alteration of the gums is reduced to a minimum, or may be quite absent. The swelling is most marked about the necks of carious and broken-down teeth; but certainly it is by no means always absent from the gums of those scorbutic patients who retain a perfect set of teeth.

If the disease be allowed to progress and to acquire an intensity which fortunately is now very rarely seen, the patient rapidly becomes weaker and more lethargic. He suffers much from shortness of breath and palpitation, and the heart's action is so weak that any muscular exertion, such as an attempt to sit up in bed, may cause fatal syncope. The muscular pains in the back and legs still persist and render him more or less helpless. There is decided emaciation and wasting of the muscles, whilst the feet and ankles become œdematous, and the face and eyelids bloated. The petechial spots and patches of ecchymoses become more livid, and make their appearance on the trunk and upper extremities. The indurated swellings increase in size and become more painful, the affected limb, usually the leg, being kept in the flexed position. The swollen gums form large, vascular growths which surround and often hide the teeth, and occasionally project from the mouth and distend the cheeks. These growths break down into large and deep ulcers, which may cause wide destruction of the gingival structure, free exposure of bone, and loosening of the teeth. No other portion of the oral mucous membrane participates in these morbid changes.

At this stage there is a general tendency to effusion of blood or sanguineous fluid. Thus a tender subperiosteal swelling—the so-called scorbutic node—may be formed in front of a long bone, most frequently the tibia; the breathing and heart's action may be suddenly disturbed by the pouring out of fluid into the pleural or the pericardial cavity; or, again, all the symptoms of pulmonary gangrene may be caused by the occurrence of hæmorrhagic foci in the lung. Scorbutic effusion into a large serous sac or into a large joint is usually of an inflammatory nature, as indicated by pain and rise of temperature. A marked peculiarity of severe scurvy is the readiness of the skin to ulcerate; not only will any existing sore suddenly thus alter its character, but an old scar, a recent wound or scratch, or even a portion of apparently sound and intact integument may become the focus of a rapidly spreading scorbutic ulcer, the characteristic feature of which is a dry black slough which, when detached, reveals sharply cut edges and a base of large livid granulations from which there is a profuse and continuous discharge of ichorous fluid. The formation of large vesicles distended by blood-stained fluid, which, according to Immermann, may result in ulceration of the skin, probably occurs only in malignant and very advanced forms of scurvy; it is very rarely, if ever, met with in the milder and ordinary forms of the disease. The tongue still remains moist, except in cases of visceral complication or extensive ulceration of the skin, but it is usually more or less swollen. There is now a tendency to diarrhœa. The stools in simple cases consist mainly of partly digested food and blood-stained fluid, but in a dysenteric patient, or in one who has been treated by strong purgatives, it may be mixed with large and abundant clots.

Notwithstanding the evident gravity of the lesions presented by scorbutic patients, and the profound morbid changes produced in almost every part of the body, this disease, in the form known to modern observers, is not only attended with remarkably small mortality, but yields at once to medical treatment and even to a suitable change in diet. Of the 790 cases admitted into the Seamen's Hospital since 1864, 15 only were fatal—a death-rate of 1·89 per cent; and of 182 received during the past seventeen years (1896-7), only one was fatal. A frequent cause of death in the forms of scurvy to which reference has hitherto been made is syncope. In many cases of death, whether during the attack or after the disappearance of most of the special scorbutic symptoms, the fatal result is due either to extreme weakness from pre-existent disease, or to a complication with dysentery, malarial fever, or some other such exhausting malady. In an uncomplicated case of scurvy, even though very severe, a supply of lime-juice and suitable vegetable food, together with rest, good nourishment, and healthy conditions, is speedily followed by the disappearance of most of the symptoms and by rapid restoration to perfect health. The external lesions usually, though not always, disappear in the following order:—first the firm subcutaneous swellings, next the swellings of the gums, and finally the petechiæ and the cutaneous ecchymoses.

A knowledge of the clinical phenomena presented in the final stage of an attack of scurvy in its worst form can only be obtained by reference to the writings of the older authors of this subject. According to Lind, it was not easy to conceive a scene of more diversified wretchedness than that beheld in the third and last period of this disease. Then the swollen legs were covered with livid and fungous ulcers; there was a profuse discharge of altered blood in the stools and urine, and also from the lungs, nose, and stomach; there was a tendency to effusions into the chest and abdomen, and towards the close of the attack there was much oppression of breathing and extreme dyspnœa; there was a troublesome cough with expectoration of fœtid and blood-stained sputa; the gums were black and gangrenous; the teeth became loose and fell out; the skin was covered by cold and clammy perspiration; there was a constant involuntary discharge of stools; the urine was retained, and the patient, unless carried off by a sudden attack of dyspnœa, gradually sank from asthenia.

Although in many of the scorbutic outbreaks recorded in the last century scurvy was often confounded with typhus and other infectious diseases, there can be no doubt that the disease itself was then attended by a very high rate of mortality. The ships of the East India Company in their voyages round the Cape often lost nearly one-half of their crew; and in Lord Anson's voyage round the globe 380 out of 510 seamen perished from the disease.

In scurvy it is difficult to draw the line between the ordinary symptoms on the one hand and the complications on the other. Formerly many lesions were regarded as specially scorbutic which were certainly due to casual and extrinsic causes; of late the tendency has been to reject even the least variable and most characteristic signs, and to reduce scurvy to a simple cachexia associated with much mental depression and muscular weakness. Thus the petechiæ are attributed to the rubbing of clothes, the swollen gums to the irritation of carious or dirty teeth, the livid patches and subcutaneous swellings to pressure and injury, and the pleural and pericardial effusions to catarrhal inflammation. There can be no doubt that, from the peculiar circumstances under which it is produced, scurvy must almost always be associated with other morbid conditions due to insufficient as well as to unsuitable food, to overcrowding, to mental depression, and to exposure to cold; as occurred in the siege of Paris, in 1871, from the failure of fuel during an exceptionally severe winter. As the late Dr. Ralfe truly asserted, simple dietetic scurvy is seldom seen, even afloat. In many instances on board ship it is really a secondary and complicated affection in men laid up from injury or some other disease, subjected to the most unfavourable hygienic conditions, and probably unable to obtain lime-juice. In such cases one would expect to find the patient suffering from diarrhœa the result of dysentery; from stomatitis the result of syphilis, or rather of its treatment; from affections of the bones and joints the result of tertiary syphilis; and from one or more fungous ulcers of the legs the result of the chronic ulceration of the lower limbs frequent in seamen. It is very doubtful whether scurvy can exert any particular influence on fractured bones. In the form now observed it never causes the absorption of old callus; and in recent fractures, though like other weakening diseases it may retard union, it is rarely, if ever, followed by a permanent pseud-arthrosis. Indeed, notwithstanding the frequent occurrence of fracture on board ship non-union is very rarely met with amongst seamen.

Much attention has been directed to the frequent association of night-blindness with scurvy. This association may occur in epidemics on land, but has been most frequently met with amongst the large crews of war-ships cruising in tropical waters. Many instances have been recorded by English and French naval surgeons in which a large proportion of men suffering from an outbreak of scurvy also suffered from night-blindness. Some of these writers go so far as to regard this disturbance of sight as a symptom of scurvy, whilst others reject the notion of any connection between the two affections. Mr. Donald Gunn, ophthalmic surgeon to the Seamen's Hospital, to whom I am indebted for much information on the subject, expresses the latest and most rational view, in stating that night-blindness has no more to do with scurvy than with any other exhausting disease, except that instances of the eye affection were first observed in scurvy patients. Night-blindness is a functional disorder depending primarily on exhaustion of the retina from prolonged exposure to bright light. Any cause that lowers the general vitality will tend to accelerate the incapacity of the retina to respond to less than the strongest stimuli. Scurvy would be the more likely to act in this indirect way, as the special conditions which give rise to it are often associated with exposure of the patient to bright light. That the retinal and not the general state is the cause is shown by—

(i.) Perfectly vigorous well-fed men, if exposed to sufficient glare, become night-blind; as in the snow-blindness of Alpine travellers, which is quite independent of the associated conjunctivitis.

(ii.) A man, however depressed by scurvy, or any other disease of malnutrition, will not show night-blindness unless he be also exposed to very bright light. It has been asserted, as the proof of the retinal, or, at any rate, functional origin of the trouble, that if one eye of a nyctalopic patient be bandaged, this eye will recover sufficiently to enable the patient to get about at night, while the other eye remains quite blind.

Blood and Urine in scurvy.—In scurvy, according to Duchek, the blood in the heart and large vessels is fluid, of a dark red colour, and contains soft ruddy clots; thus resembling the blood in enteric fever. In anæmic bodies, after long protraction of the disease and extensive hæmorrhages, it is lighter in colour, but still coagulable. When taken from a living scorbutic patient it differs but slightly from healthy blood. Microscopical examination has failed to reveal any definite change in it. Hayem found in blood taken during life that the number of white globules was normal, and that there was no alteration in the appearance of the red globules. On the other hand, Laboulbène found the number of white globules or leucocytes considerably increased,—a condition, however, which he considered of no special importance with regard to scurvy, as it is observed in many other pathological conditions. The statement of Mr. Busk, in 1841, that the amount of fibrin in scorbutic blood is increased, though opposed by Andral on the strength of a very doubtful observation of scurvy, and afterwards by Becquerel and Rodier, has been fully confirmed by more recent observers. Chalvet, who has made very careful analyses of blood taken from scorbutic patients, agrees with Busk that the blood globules are diminished and the amount of albumin increased. As a result, no doubt, of the impossibility of obtaining sufficient quantities of blood for such purpose, no endeavour has been made to determine the relative quantities of the different inorganic constituents. The assertion of Becquerel and Rodier, that there is an increase of chloride of sodium and other salts in the serum of the blood, has been disproved by the later investigations of German chemists.

Hæmaturia seldom occurs in scurvy, even in the severe cases. The urine during the course of the scorbutic attack is scanty, dark-coloured, clouded, and in severe cases from time to time slightly albuminous. As the symptoms pass off, and the patient becomes convalescent, it increases in quantity and becomes paler. The specific gravity increases during the attack, and decreases after it. In correspondence with these changes Duchek found a decrease of the solid constituents, except phosphoric acid and potash, in the first stage; and subsequently a restored relation between all the solid elements. In a more recent investigation, to which allusion has already been made, Dr. Ralfe found in the urine of scurvy patients (i.) an increase of uric acid; (ii.) a diminution of the acidity of the urine; and (iii.) a reduction of the alkaline phosphates.

The following are the *complications* most frequently observed in scurvy:—(*a*) Inflammatory effusion in the pleural cavities; (*b*) Pneumonia and gangrene of the lungs. These affections were very prevalent amongst scorbutic patients in the Crimea. It would have been interesting to trace their association with ulceration of the gums, as the excellent descriptions of the pulmonary symptoms given by Haspel and Buzzard seem to indicate an infective rather than a catarrhal origin of these lesions. (*c*) Pericarditis with abundance of sanguinolent effusion. (*d*) Diarrhœa usually of the simple irritative form, but in severe land epidemics and amongst seamen, often of hæmorrhagic character, in consequence of the presence of dysentery. (*e*) Dropsy: dropsical œdema of the foot and ankle is a very frequent complication; rapid effusion into the whole of the lower limb on one side was occasionally observed during the epidemic at the siege of Paris. Ascites rarely occurs, and when present is probably the result of renal or hepatic disease. Hydrothorax and hydrarthrosis are not infrequently met with.

In scurvy there is not, as is generally supposed, any marked tendency to bleeding from internal organs. Epistaxis occurs more frequently than any other form of hæmorrhage; melæna is met with occasionally as a result of dysenteric ulceration or of the action of strong medicine; hæmaturia and hæmoptysis occur very rarely.

Pathological anatomy.—Our knowledge of the pathological changes produced by scurvy still remains very imperfect, notwithstanding the researches of Duchek and the careful observations made by French pathologists in 1871. One important point, that has been well established by the latter, is the exemption of the blood-vessels from morbid change. Another point which, if confirmed by further investigations, will also prove no less important, is the observation made by Leven of a general fatty degeneration of the organs. According to this writer, the striated fibres of the voluntary muscles, and of the muscles of the heart, are destroyed, and are replaced by fatty granulations. This fatty degeneration also invades other organs, such as the kidney, the liver, and the lungs; the blood-vessels alone remaining free. It is very probable, however, that these changes, which have escaped the notice of many competent pathologists, are due to cachectic or other general morbid conditions associated with scurvy but not dependent on it.

The condition of the body after death from scurvy is such as might be expected in a case of cachectic disease marked by a tendency to hæmorrhagic effusion. An interesting fact, to which attention has often been directed, is that, except in protracted and very severe cases, there is very little wasting of the subcutaneous fat and the muscles. Decomposition sets in rapidly, and the petechiæ and ecchymotic patches observed in the skin during life are soon obscured by post-mortem lividity. The subcutaneous tissue, especially in the lower limbs, is suffused by blood-stained fluid, and here and there are collections, varying in extent, of effused blood, some quite black and others of a paler colour, from cherry-red to yellow. In the indurated swellings, such as those so often met with at the back of the thigh and knee, the muscles and tendons will be found embedded in a thick and firm clot, and the muscles within their sheaths studded with hæmorrhagic foci, which, like the extra-muscular effusions, are soft and ruddy when recent, and pale, tough, and scar-like when of long standing. Similar deposits, though of much less extent, may be found in connection with bones, in most instances the tibia, just beneath the periosteum; and also, as Immermann asserts, within the bone, especially in the midst of spongy tissue. Effusions are sometimes found between an epiphysis and the shaft of a long bone in a young subject; and also between the ribs and their cartilages. Many of these effusions do not consist merely of altered blood or sanguineous fluid, but of a fibrinous and plastic material which, after a time, is traversed by minute vessels which may be readily injected. In most cases of mild and uncomplicated scurvy the viscera present but few morbid appearances. The lungs, except at their lower lobes, are usually collapsed, pale and anæmic. The cavities of the heart are sometimes empty; at other times they are distended by dark-coloured blood containing soft and gelatinous clots. The organ itself, as a rule, is small and flabby. In many cases the only marked indications of a scorbutic taint are hæmorrhagic spots scattered over the pleura and over the roots of the large vessels of the heart. The changes observed in cases of grave inflammatory lesions which may involve the pulmonary organs in scurvy have been fully described by Buzzard. Of these the most considerable are complete engorgement of the lungs; a diffusion of ecchymotic deposits which compress and obliterate little by little the pulmonary tissue, and often form large fluctuating tumours composed of fluid blood and gangrene. Many medical men versed in severe scorbutic attacks have made mention of effusions of more or less blood-stained serum into the pleural and pericardial sacs. Such effusions, it seems, take place rapidly, are generally abundant, and always associated with fever and other indications of inflammation. Mention has been made also of similar effusions, which, however, are not so frequent, into the abdominal cavity, together with ecchymotic spots and patches on both the parietal and the visceral peritoneum. The abdominal lesions observed in seamen are usually such as are due to tropical diseases; to dysenteric ulceration or pigmentation of the large intestine; a swollen spleen; and a swollen and an engorged liver. Although transient albuminuria is not of infrequent occurrence in scurvy, no constant morbid change nor any special scorbutic lesions have been observed in the kidney. All pathologists agree as to the rarity of any intracranial lesions in scurvy. It has been pointed out by Dr. Buzzard that, considering the delicate structure of the brain, it is remarkable that scorbutic lesions occur by no means so commonly in this organ as in other and less vital parts of the economy. Here clinical and pathological data are in correspondence; as in even the most severe cases of scurvy the intellect remains clear to the last. In considering the few disease, there are well-marked distinctions between the two affections. Purpura is not due to any special defect in diet, nor is it relieved by an increased supply of antiscorbutics. It is characterised by plethora rather than anæmia, and shows a marked tendency to epistaxis and bleeding from internal organs; it affects chiefly the mucous membrane and the skin, whilst the muscles, bones, and subcutaneous soft parts remain free. There is no swelling of the gums. The ecchymotic spots and patches are more vivid in colour and more generally diffused than those of scurvy.

It would hardly be possible to mistake sporadic scurvy for hæmophilia; the latter being a chronic affection of a congenital and hereditary character, met with usually in young subjects, and presenting signs of hæmorrhage from time to time, usually after injury. Three other hæmorrhagic affections have in the diagnosis of scurvy to be taken into consideration. These are leucocythæmia, splenic anæmia, and pernicious anæmia, which affections, like scurvy, are preceded by cachectic conditions. Leucocythæmia, indeed, in some rare instances, presents hæmorrhagic swelling of the gums. Scurvy would, however, be set aside at once by the presence of glandular swellings, of splenic enlargement, and of definite excess of leucocytes in the blood. With regard to pernicious anæmia, which resembles scurvy in many respects, the distinction should rest upon the chronic course of the former disease, the absence of any special dietetic fault, a marked difference between the waxy pallor of those affected and the sallow hue of the scorbutic subject, and the examination of the blood (art. p. 408). [For "Splenic Anæmia," *vide* p. 539; "Infantile Scurvy," p. 604.]

Prognosis.—The prognosis of a case of scurvy is favourable if the attack have not lasted long, if there be no visceral complications, and if the patient can be supplied at once with efficient antiscorbutics and placed under other good hygienic conditions. In cases of prolonged scurvy death may occur from prostration and general loss of power. Abundant inflammatory effusion into the pericardial or pleural sacs must be regarded as serious; although, as a rule, such effusions disappear with remarkable rapidity under the influence of an improved and antiscorbutic diet. Dysentery is a serious complication; if it do not lead to a fatal result it will certainly retard convalescence. Notwithstanding the low mortality that has attended scurvy during the present century, care should be taken in every case not to give too favourable an opinion; as, even under the most promising conditions, and at any moment in consequence of a moderate muscular effort,—such, for instance, as that of sitting up in bed,—fatal syncope may occur. Persistence of a normal temperature, a tendency in the hæmorrhagic spots and patches to fade, an increased flow of urine, and a clean tongue are all to be regarded as favourable signs. On the other hand, scanty and high-coloured urine, an increased tendency to local hæmorrhages, an occasional elevation of temperature, diarrhœa, difficulty in breathing, signs of cardiac failure, are all to be regarded as indications of the steady persistence of the scorbutic attack.

Prevention.—If, as has been concluded, scurvy is invariably caused by a much diminished supply or a total absence of vegetable food, then the means for the prevention of the disease must consist mainly in correcting this fault, and in ensuring a full and mixed diet. On land this question, which, except in children, very rarely presents itself in times of peace and plenty, becomes one of pressing urgency in war; and then forms one of the greatest difficulties in military hygiene. In long sea-voyages it is always necessary to take the matter into consideration, and to endeavour to make good the enforced deficiency of fresh vegetable food by the supply of preserved vegetables and fruits, and of some antiscorbutic preparation. Of these substitutes for natural nutriment the former, though not the more efficient, are certainly the more convenient and trustworthy. Lime-juice, when taken day after day, becomes distasteful, and often disagrees with those who take it; as it is not an article of food there is no certainty of its being regularly consumed except under such conditions of discipline as exist in the Royal Navy and in large mail steamers: moreover, in spite of all precautions it may deteriorate after long storage on board ship. Notwithstanding the improved means of preserving vegetables, and the undoubted value, as antiscorbutics, of preserved potatoes, cabbages, carrots, and so forth, they are still much inferior in this respect to vegetables and fruits that are quite fresh. For this reason short voyages are an important factor in the prevention of scurvy. Dr. Curnow, alluding to the decrease of scurvy in merchant ships in correspondence with the increase of steam shipping and the decline in the number of sailing vessels, justly points out that more rapid voyages mean more frequent supplies of fresh food, and thus eventually lead to the practical extinction of this disease.

Whenever the ship touches at a port no opportunity should be neglected of supplying the crew with fruits and fresh vegetables, and in taking an abundance of these on board. A good supply of preserved vegetables and of lime-juice on board ship would not justify any neglect of this precaution.

To the above-mentioned antiscorbutics, which are strictly of vegetable origin, may be added milk, which contains all the elements required for the nourishment of the body; and also certain beverages such as malt liquors and light wines, especially claret, tea, and very probably cider which was regarded by Lind as the best of all. These beverages are remarkable for the large quantity of potash which they contain in combination with organic acids. Alcohol not only fails to act as an antiscorbutic, but, when taken freely, is undoubtedly an active contingent cause of the disease.

It is necessary, also, to take into consideration the means by which, in the absence of efficient antiscorbutics, an outbreak of scurvy may be averted. These should consist in removing, as far as possible, all other conditions that interfere with the maintenance of good health; in an endeavour to supply good and nutritious food, particularly fresh or well-preserved meat; to avoid exposures to extreme heat and cold; to promote moderate but not excessive exercise; to afford suitable clothing, and to maintain good ventilation and other favourable hygienic conditions.

Treatment.—The treatment of scurvy, provided the attack be free from complication, is very simple. The chief indications to observe are the supply of those elements of food which have hitherto been wanting, to restore strength and vigour, and to relieve the more severe local lesions. In most cases all these indications may be fulfilled by strictly dietetic means; the scorbutic taint being removed by the free use of fresh vegetables, and the general weakness overcome by nourishing and readily assimilable food. Under such treatment the more serious symptoms, such as those of pleural and pericardial effusions, will, in most cases, disappear together with those that are less grave and more superficial. In dealing with a scorbutic patient care must be taken to avoid such articles of diet as might intensify any complicating affection, particularly dysentery; and, in the second place, by keeping the patient in the recumbent position, to prevent syncope or sudden death. The local lesions, even the most severe, usually disappear with singular rapidity, and by the end of the second week the patient may be restored to his former state of health. The diet should consist of a free supply of fresh vegetable (potatoes, green vegetables) with oranges and other succulent fruits, and eggs, fresh milk, strong soups, and beef tea; to these, as the health improves and the digestive organs become stronger, may be added chicken and lean meat. There is no need, as a rule, to give medicine; nor, if a good supply of vegetables be at hand, even lime or lemon juice. In complicated cases only will it be found necessary to resort to medicinal treatment. Dysenteric diarrhœa must be met by appropriate remedies; and in a case of extensive pleural or pericardial effusion it may be advisable to remove the fluid by aspiration. The gums, if much swollen and ulcerated, should be touched with solid nitrate of silver or sulphate of copper; or be brushed over with a solution of one part of chromic acid in five parts of water. If there be any scorbutic ulceration, the swollen and sloughing sore should be douched three or four times a day with cold sterilised water, or salt and water, and be dressed in the intervals with some iron lotion, or boracic ointment. The petechiæ will disappear rapidly and need no local treatment. The indurations, if they remain tender and show but little tendency to diminish, should be treated by gentle massage and the compression of a flannel bandage.

W. JOHNSON SMITH.

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W. J. S.

INFANTILE SCURVY

SYN.—*Scurvy Rickets.*

Definition.—The scurvy of childhood, like that met with in adults, is a morbid condition of blood and tissues due to defect of diet. It is characterised by great and progressive anæmia, tendency to syncope, cachectic earthy complexion, marked muscular debility, mental apathy and depression, sponginess of gums, and hæmorrhages into various structures, notably under the skin and periosteum and into the muscles, especially of the lower limbs.

The disease has a definite dependence upon the privation of fresh food: in the case of adults usually of fresh meat and fresh vegetables; in the case of infants of fresh milk or other fresh food which supplies the same antiscorbutic property; in both it is immediately relieved and rapidly cured by the administration of the fresh elements which have been wanting.

History.—The existence of scurvy in young children, in sporadic form, apart from its occurrence in common with adults under the special conditions of epidemics, has only been recognised within a comparatively recent period. It appears from the researches of Dr. Barlow that isolated cases of similar character had been observed and recorded in Germany from the year 1859 to 1873, by Mohler, Bohn, Hirschspring, and Senator, as examples of acute rickets. The first suggestion of their real nature seems to have been made by Dr. Ingelev, a Swedish physician, in recording ably quiet and contented when left at rest and undisturbed by movement or pressure. The legs especially are tender; the child no longer kicks them about, but keeps them drawn up and still. As the condition advances, the tenderness becomes extreme; the child screams out, not only on the least movement, but on the approach of a hand to touch it; and the lower limbs lie splayed out, and absolutely motionless, as if paralysed. This extreme dread of touch and movement, this quasi-paralytic stillness of the limbs are together almost diagnostic. On examining the legs, swelling of the periosteum will probably be found along the shaft of one or both tibiæ above the ankle; the thigh bones may be affected in the same way, and there may be œdema of the dorsum of the foot. In some instances this periosteal swelling is slight and not apparent at first sight; in others it is palpable and at once attracts observation. With the subperiosteal changes are associated, in severe cases, deep-seated hæmorrhages into the muscles themselves, causing puffy swellings and brawny indurations. In certain cases these are so great as to excite the suspicion of abscess, and they have led more than once to surgical exploration, which has, however, revealed nothing but blood-clot. There is no heat or redness of the surface, no sense of fluctuation, and no rise of body temperature: the temperature is normal or subnormal, except in a few instances where the hæmorrhages are large and recent, when it may run up to 100° to 102° for a period of a few days.

The upper limbs may be unaffected, but there is often some swelling and tenderness of the forearm above the wrist; and more rarely on the humerus. Occasionally other bones are invaded; similar swellings have been observed on the ribs, on the scapulæ, and on the skull. In one instance under my own care the chief periosteal swelling was on the malar bone. The joints proper escape, although periosteal changes near the epiphyses cause a fulness just above them, which at first sight appears to be connected with them, and is not infrequently mistaken for that of rheumatic arthritis. At these joints also, occasionally, crepitus due to separation of the epiphysis from the shaft may be detected; or more rarely fracture of the shaft itself. In some instances the same fractures are found close to the juncture of the ribs with the cartilages, giving rise to a curious depression of the sternum and costal cartilages connected with it, as if it had been driven forcibly inwards towards the vertebral column. A similar deformity is sometimes observed in rickets without known scurvy; in such cases there is no fracture of the rib bones, but acute bending only.

As these signs of affections of bones and periosteum are manifested, the anæmia progresses also, and the complexion assumes the sallow earthy hue so characteristic of true scurvy, due probably to diffusion or deposit of altered hæmoglobin. With this the debility increases, the patient becomes more limp of body and weak of back, and cardiac power grows more impaired. Other characteristic signs also begin to appear, notably the one which is really pathognomonic; namely, sponginess of the gums.

Spongy gums are swollen, soft, boggy, purple, hæmorrhagic. In severe cases, when the teeth have come through, they become so swollen as to protrude between the lips in livid bleeding lobulated masses, sometimes so large as to hide the teeth altogether. These spongy excrescences bleed freely, soon begin to ulcerate, and exhale the horribly putrid odour met with in sea scurvy. The teeth become loose and frequently fall out. In some instances the gums are so tender that a child can only be made to take food with difficulty. The change in the gums is not, however, invariably present. If the incisors have appeared there is almost always some purple discoloration to be seen at their base. If the teeth have not appeared, but are approaching the surface, the same purple colour is found over the gums which cover them, or minute ecchymoses appear there. As Dr. Barlow has aptly pointed out, the manifestations of this condition of the gums depends not upon the severity of the disease only; it has also a definite relation to the number of the teeth; and that this is strictly analogous to the sea scurvy of adults, in which disease where the teeth have fallen out the portion of gum in relation to them does not become spongy, although in that portion in relation to existing teeth the condition is fully established. To undergo this change the gum must be in functional relation to the teeth. Another marked and characteristic feature in severe cases of infantile scurvy, as in that of adults, is the supervention of hæmorrhages of various kinds into different tissues and organs; in some cases as petechial spots on the skin, occasionally as larger subcutaneous hæmorrhages, especially on the trunk and lower extremities; sometimes there is extravasation into the loose areolar tissue below the lower eyelid. In one case under my own care the supervention of a well-marked black eye, which suddenly followed a fit of crying, decided a previously doubtful diagnosis. The fragility of the capillaries in these cases is further illustrated by the readiness with which discoloration and bruise marks are produced upon the skin by slight injury; such as the pressure of handling or the blow of a slight fall.

Occasionally a curious phenomenon appears in the shape of sudden proptosis of one eye, with slight discoloration of the upper eyelid and redness of the conjunctiva, due to hæmorrhage under the periosteum of the orbit. The hæmorrhagic tendency of the disease is further marked in some cases by hæmaturia, which is occasionally the first or the principal sign of the supervention of the scorbutic state. In addition there may be epistaxis, or hæmorrhage from the bowel; but these occurrences are not common, and the loss of blood is seldom or never very copious. In some of the slighter cases the symptoms of the disease may be limited to one or two signs, the significance of which, when occurring alone, may be overlooked or misinterpreted. In some instances hæmaturia may be almost the only manifestation in addition to anæmia and muscular debility; in others orbital hæmorrhage and proptosis may be the only prominent features. More frequently, perhaps, there is merely slight purple staining over the gums of the erupted or pushing teeth, with some tenderness of limbs and general anæmia and cachexia.

FIG. I.

INFANTILE SCURVY

Showing spongy gums, proptosis of right eye, external strabismus due to post-orbital hæmorrhage, with ecchymosis and œdema of right upper eyelid.

H. D., æt. 9 months. Sole diet for previous six months, condensed milk and malted farinaceous food. Drawn from life, July 26, 1895.

FIG. I.

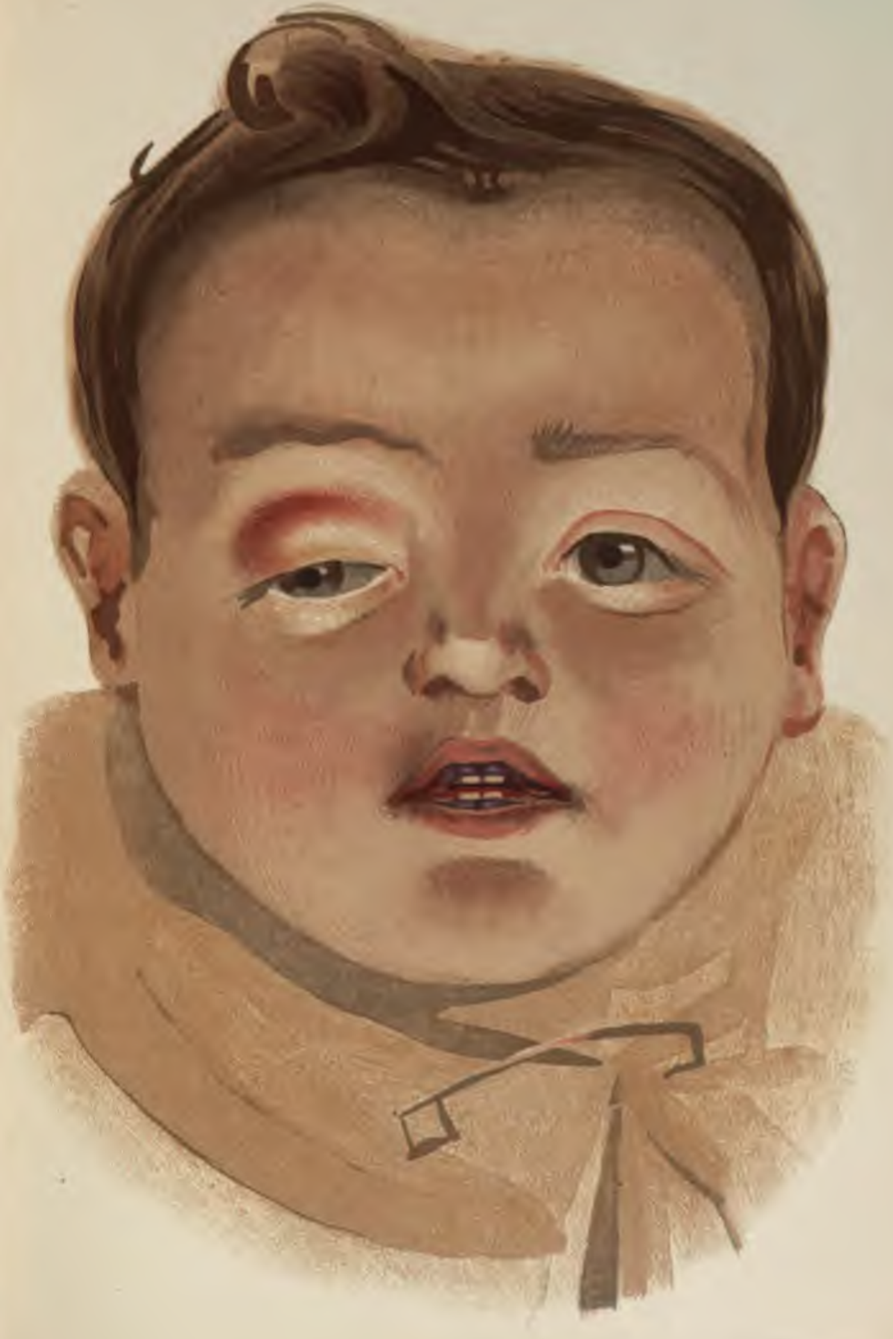

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FIG. II.

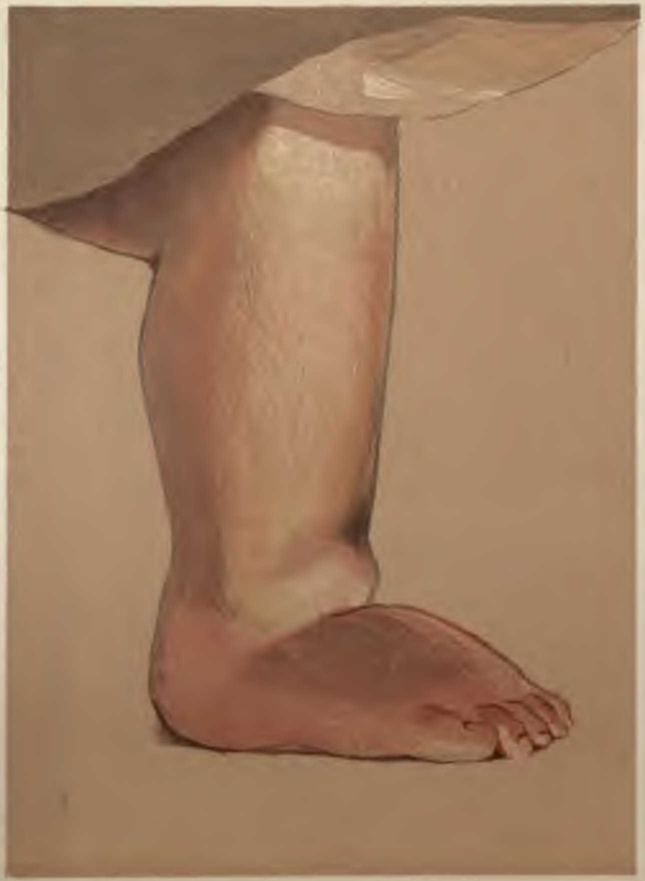

FIG. III.

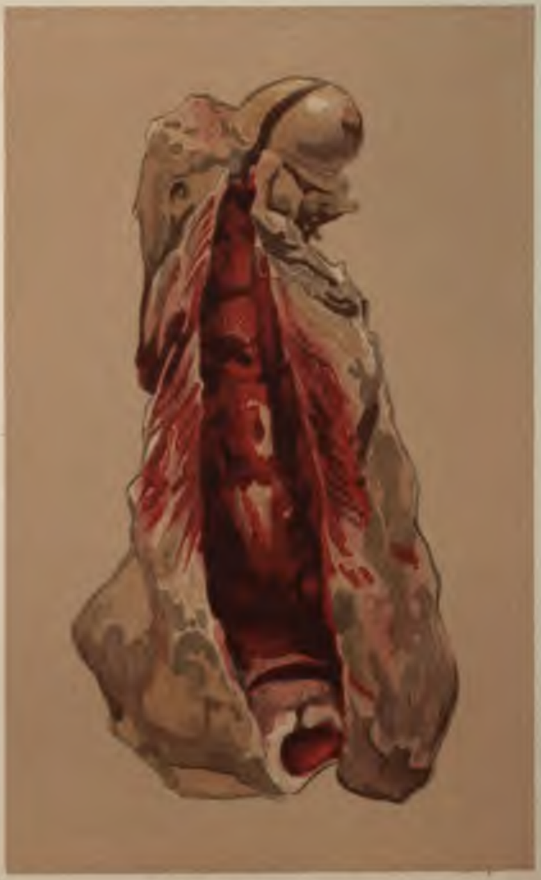

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FIG. II.

INFANTILE SCURVY

Showing swelling in front aspect of tibia from subperiosteal hæmorrhage, just above the ankle, with œdema of ankle and foot.

From the same patient as Fig. I. Drawn from life, July 26, 1895.

FIG. III.

Post-mortem appearances presented by the femur and surrounding tissues in a fatal case of infantile scurvy, namely, hæmorrhages and masses of blood-clot under the periosteum, which is vascular and thickened; hæmorrhage and serous infiltration into the deep muscles adjacent; hæmorrhage into the central canal of the bone, and fracture of the shaft near the epiphysis.

Sketched from a preparation in the Museum of the Hospital for Sick Children, Great Ormond Street. From a case of Dr. Barlow's.

There are no doubt cases slighter still, where the only symptoms are tenderness of limbs evidenced by irritability and intolerance of handling, with perhaps some signs of rickets, to which the symptoms are referred. The fact that in many instances these conditions, so closely associated with scurvy, rapidly disappear upon antiscorbutic diet, while those more particularly identified with rickets are little affected by it, is very suggestive of their real nature.

This characteristic group of symptoms, in well-marked examples of infantile scurvy, accurately correspond, not merely individually but as a composite whole, with the series of phenomena met with in the epidemic form of the disease. The pallid, earthy complexion, the progressive anæmia, the excessive muscular feebleness, the tendency to syncope, the various hæmorrhages and their seat, the hæmaturia and albuminuria, the œdema, the swellings of periosteum and of muscle, the extreme tenderness of limb, the special implication of the lower extremities are the same in both. The fact that the bones suffer somewhat more severely in proportion to other tissues is explained by the great formative activity which pervades these parts in infancy. The symptoms are indeed practically identical with those of the adult with, in most cases, the signs of an underlying rickets; such as beading of the ribs, enlargement of the epiphyses, head-sweats, and laryngismus. In most instances, however, these signs are slight, and in some no indication of rickets can be discovered.

Course of the disease.—The course of the disease varies according to the degree of its intensity and development, and the conditions under which it arises and under which the patient remains. If the defects of diet in which it has its origin continue unchanged, and the hygienic surroundings are unfavourable, the patient grows steadily worse, the debility and anæmia increase and become extreme, and the cachexia profound. In this state the child may die suddenly from hæmorrhage into some vital organ, or from syncope, or from more gradual exhaustion; or from some intercurrent affection such as bronchitis or pneumonia, or diarrhœa; or again, an acute infectious disease may prove rapidly fatal to the enfeebled organism. Occasionally, without special treatment, slow amelioration of the disease takes place after a time; some change of food in the ordinary advance to a wider and more varied dietary, as the child grows older, leads to a gradual improvement in the condition, and the symptoms after some months may disappear. Relapses often occur; and in any case the disease, when uninfluenced by treatment, runs a chronic and protracted course, unless cut short by some fatal accident or complication. If, however, the nature of the affection is recognised, and proper antiscorbutic treatment adopted, improvement is immediate, and recovery so marvellously rapid that the child may be practically well in from two to three weeks. The swelling of the limbs subsides, tenderness and the dread of movement disappear, the child begins to move its limbs again voluntarily and to sit up once more, the hæmorrhages cease, and the anæmia and cachexia and asthenia quickly decline.

Some wasting of the muscles of the affected limbs remains, and for some time afterwards hard thickening can be felt round the shafts of the affected bones. If fractures have occurred, they are repaired without obvious deformity, except in rare cases, when they take place in the middle of the shaft of a long bone; then much thickening may remain for a time. In the end, however, the recovery is final and complete; although, where accompanying rickets exists, the signs of this condition may long persist. As already observed, however, the rachitic complication in these cases is usually slight, and but rarely severe of degree.

Morbid anatomy.—For an accurate knowledge of the morbid changes which are associated with the signs and symptoms described, we are chiefly indebted to the careful investigations of Dr. Barlow, who, in a paper published in the *Medical and Chirurgical Transactions* for 1883, has given an exhaustive account of the appearances met with after death, and has shown conclusively that in this respect also the conditions observed are identical with those found in the true epidemic or sea scurvy of adults. These conditions have been further examined and confirmed by other observers in this country and abroad, and again by Dr. Barlow himself, who has set forth the results in the Bradshaw Lecture for 1894. The details of morbid changes given below are largely drawn from this source.

As will have been gathered from the previous account of the symptoms, the principal lesions found after death are due to increased vascularity and extravasations of blood into various tissues. The most extensive and important of these are found in connection with the periosteum, the bones, and the muscles. These changes are most common and extreme in the lower limbs; but they are met with also, although less frequently and in minor degree, in connection with the bones of the upper extremities, and of the skull.

The periosteum of the long bones of the leg and thigh is highly vascular, and blood is effused more or less extensively round the shaft beneath it, detaching it from the bone and forming a thick sheath of blood-clot between periosteum and shaft; the tibia and femur are usually most affected in this way (Fig. II. of Plate). The extravasations correspond with and account for the exquisitely tender and sensitive swellings observed during life. In some cases similar hæmorrhages occur under the periosteum of the humerus, of the scapula, of the ribs, and of the cranial bones, corresponding to the swellings described there. One of the most characteristic of these, when it occurs, is the extravasation into the loose tissue which connects the roof of the orbit with its periosteum, and accounts for the curious proptosis which has been described in some cases, the eyeball being thus pushed downwards and forwards. Hæmorrhage is also found sometimes in the loose tissue of the upper and lower eyelids, causing the black eye of which mention has been made. A thin layer of newly-formed osseous material is occasionally found beneath the upraised periosteum, forming a bony sheath round the shaft of the long bones, or a similar formation of delicate bony film under the periosteum of the flat bones, such as the scapula. Hæmorrhages also take place, in some cases, into the medullary cavity of the long bones of the limbs and of the ribs, forming masses of blood-clot there; the medulla itself being soft and reddened. The muscular swellings (Fig. III. of Plate) are due to deep-seated extravasations, especially in the muscles of the lower limbs, which are also sodden by serous effusion, wasted, flabby and pale. In rare instances hæmorrhages have been met with in some of the joints, and under the dura mater of the skull; and the purpuric blotches and bruises which are liable to follow handling are also, of course, hæmorrhagic in nature. Similarly, extravasations have been observed in the pleura, the lungs, spleen, intestines, kidney, and mesenteric glands. In one fatal case under my care, in addition to the spongy, bleeding gums, there were extensive hæmorrhages into the lung, and smaller extravasations and ecchymoses into the intestinal mucous membrane and into the lymphatic glands; the bones and muscles being free. Similar cases have been observed by others. When hæmorrhage into the central canal of the long bones occurs, the bone itself suffers so that the compact tissue of its wall becomes absorbed and rarefied, and is reduced to a thin shell. A similar condition is found in the ribs.

Another characteristic feature of the morbid changes in the bones in scurvy is the occurrence of the fractures before alluded to. These take place especially in the rarefied imperfectly ossified portion of the long bones connecting the shafts with the epiphysis, and sometimes a little above this; the two extremities of the femur and the upper end of the tibia are the most frequently affected in this way; occasionally the upper end of the humerus shows a similar fracture. The ribs again, as previously noted, are occasionally broken away from the costal cartilages. The fractures are due in part to the weakening of the shaft by the detachment of the periosteum by the hæmorrhage into the medullary canal, and by the extensive absorption of the trabecular structure.

In the mouth the gums are seen to be spongy, swollen, and sodden with serum; and perhaps clotted with blood. The teeth, if present, may be loose and on the point of falling out.

The viscera show no morbid changes beyond those caused by the hæmorrhages which have been detailed, and the well-marked anæmia. The muscles likewise are anæmic, soft, and wasted, while those of the limbs most affected usually show the local hæmorrhages so often alluded to. The blood is watery. In the majority of cases, but not in all, the bone changes of rickets are found in addition to those of scurvy.

It will be seen that, in like manner with the symptoms observed during life, the morbid changes discovered after death in infantile scurvy, namely, the various hæmorrhages and their seat, the rarefaction of bones, the fractures, the formation of bony plates under the periosteum, differ in no respect from the similar changes found in the epidemic scurvy of adults.

Etiology and general pathology.—It will be gathered from what has gone before that the general pathology of infantile scurvy, occurring sporadically, is, in its symptoms and morbid anatomy, in all essential points the same as that of the scurvy of adults. The original view of the earlier observers in Germany that this affection is an acute form of rickets has proved erroneous, and is generally abandoned. It was based upon an imperfect acquaintance with the morbid anatomy of the disease, as well as of the exact dietetic conditions under which it arises. Although, as has been stated, a certain degree of rickets is usually present, this is not a constant and invariable accompaniment; there is no relative correspondence or proportion between the degree of rickets and the degree of scurvy, nor indeed between it and the supervention of scurvy at all. In severe and advanced cases of rickets where the bone-changes are extreme and there is marked cachexia, with head-sweats, laryngismus, and all the signs of severe and progressive disease, the gums are not spongy, there are no subperiosteal hæmorrhages, no muscular or subcutaneous extravasations, no hæmaturia, no hæmorrhages elsewhere. Rickets is not in itself hæmorrhagic in any degree, so that the scorbutic features are not a mere manifestation of severe or acute rickets. Moreover, signs of rachitic implications may be altogether absent, as in two cases under my own observation recently, and in a similar instance, recorded by Dr. Northrup of New York, in which no rickety change of any kind could be detected on post-mortem examination.

It had been thought possible that the condition might be one of purpura hæmorrhagica, or hæmophilia concurring with rickets. The lesions found after death and the course of the disease, however, are widely different, and no family history of hæmophilia can be traced. Moreover, as has been shown above, the underlying basis of rickets is not always present. Again, the disease is not simply the purpuric state which is liable to supervene in the late stages of wasting disease, for the subjects of it are not merely not marasmic but in some instances fat and full tissued; nor is it the hæmorrhagic stage of lymphadenoma or leukæmia, for there is no enlargement of lymphatic glands or spleen; nor is it a phase of congenital syphilis, the signs and history of which have been wanting in all the cases seen by myself, although some instances have been recorded in which this condition was concurrent. The evidence of the real nature of the disease is completed by the effect of full antiscorbutic treatment, and this, added to that drawn from the dietaries of the children affected, and the pathological changes found after death, is conclusive. There is nothing in the whole range of medicine, not even excepting the effect of thyroid extract in myxœdema, more striking and remarkable than the immediate and rapid recovery which follows the administration of fresh vegetable material and other fresh elements of food in these cases of infantile scurvy. Simple rickets is no doubt influenced by dietetic treatment, but it is not especially influenced by antiscorbutics; moreover, the effect of diet is gradual and follows slowly, in marked contrast to the instant and immediate amelioration which follows in the case of scurvy. Lastly, diet is powerless to arrest the hæmorrhages of purpura and hæmophilia, or those of lymphadenoma or leukæmia.

Sporadic infantile scurvy, then, like the epidemic affection, the so-called true or sea scurvy, consists essentially in an altered and depraved condition of blood, which gives rise to an enfeebled and fragile state of the capillaries, so that serum readily transudes and the vascular wall easily ruptures. Hence follow the serous infiltrations, fibrinous exudations, and hæmorrhages which have been described. The exact nature of this defect in the blood which is the immediate cause of the softness, permeability, and fragility of the capillary walls, has not been ascertained with certainty. It would appear, however, from the researches of Busk, Garrod, Ralfe, and others, that the alkalinity of the blood is diminished; probably because neutral salts such as the chlorides are increased at the expense of the alkaline salts, or else that the latter are absolutely and not only relatively diminished. The defective alkalinity leads to dissolution of the blood corpuscles, ecchymoses, and fatty degeneration of muscles and secreting cells. The source of the defect in the blood has been clearly traced to lack of fresh food and notably of fresh vegetable food.

Whatsoever the exact nature of the antiscorbutic element, it is clearly supplied by this kind of food, and the lack of it sets up the disease. The scorbutic state arises under conditions of life which involve such privation; as on long voyages, expeditions, shipwrecks, the campaigns of armies, sieges, or famines: and it is intensified and fostered by conditions of malhygiene, by hardship, exposure, foul air, want of light, and probably also by the prolonged use of salt provisions.

In the case of children the cause has been traced with equal certainty to this deficiency of the fresh element in food. The natural ordinary food of infants is milk alone. Instances of children becoming scorbutic when at the breast are limited to epidemic scurvy, and no case of the kind has ever come within my own cognisance.

With the exception of one or two doubtful cases, of which the details of breast feeding and diet are imperfectly given, the only instances of scurvy arising in sucklings are those when the nursing mother has been suffering from scurvy at the time. Similarly no case has come under my observation in which scurvy supervened on an ordinary diet of fresh cows' milk unaltered by peptonisation, or by the prolonged heating of a sterilising process.

Fresh milk must necessarily contain the antiscorbutic element, whatever the exact nature of this element may be; for milk is the source from which it is supplied to the infant organism. A careful examination of the conditions of diet in a large number of cases confirms this inference, and establishes the prime fact that the children who become affected with scurvy have been brought up upon a diet deficient in fresh milk. In 37 cases under my immediate observation, in which the details of feeding could be ascertained with exactness, it was found that in the great majority—namely, in 27—no fresh milk at all had been given for a long time before the attack. In the majority of these none had been given at any time; and in the rest only at the commencement of hand-feeding, having been quickly and finally abandoned because it did not agree. In the 10 remaining cases a very small quantity of fresh milk had been given; in 4 of these, however, for a few weeks only, the children having been previously brought up entirely on dried or patent foods. In 2 cases only out of the whole number of 37 had the defect of diet been in any degree compensated by the addition of fresh elements in the form of a small quantity of raw meat juice. In a few cases the food was entirely limited to some dried farinaceous preparation made with water only. In the greater number of instances, however, the scorbutic condition arose upon an exclusive diet of one or other of the proprietary preserved foods, consisting of malted flour mixed with dried animal matter, and prepared by the simple addition of water, without fresh element in any form.

Next to these in frequency come the cases where the diet has been restricted for a considerable period to one of the predigested foods, more particularly the pancreatised farinaceous foods, in which the milk added is pancreatised in the process of preparation; or upon a prolonged diet of peptonised milk, especially peptonised condensed milk. Simple condensed milk is responsible for a certain number of cases. In a larger number still, however, the disease had arisen after the continued use of the commercial preparation of so-called "humanised" milk—that is, milk deprived of a portion of its casein and sterilised by heat or other methods to make it keep.

It is clear that the process of peptonisation or pancreatisation of milk greatly impairs its antiscorbutic property; and this is also the unmistakable result of prolonged heating at high temperatures, as in the process of preparing condensed or desiccated milk. Although no cases of scurvy arising upon a diet of simple sterilised milk have actually come under my notice, it is highly probable that its antiscorbutic virtue is lessened by the process; and leading physicians both in Germany and America, where it is more largely used than in this country, deprecate on this ground permanent feeding on milk sterilised in this way. The mere raising of milk to the boiling-point for a few moments appears to have no serious deteriorating influence, although it is probable that its antiscorbutic power is lessened in some small degree by this minor process.

The antiscorbutic power even of fresh untreated milk is comparatively feeble, far less than that of fresh vegetables; and it probably varies to some extent according as the animal from which it is drawn is fed on dry food, or grass or roots. The imperfect power of milk in this respect was long ago noted by Dr. Parkes, who investigated the point; his conclusion was generally that in the case of adults one pint to one pint and a half was not always sufficient to prevent scurvy in the absence of fresh vegetable food.

The relatively slight antiscorbutic virtue of milk is further exemplified by its slow and imperfect curative power when used as an antiscorbutic agent in the treatment of scurvy. To be effective it must be given in large quantity. This fact seems to explain the occasional occurrence of the disease in children who have milk in small amount, and the deleterious effect of any impairment of its properties by peptonisation or over-heating. In addition to the cases which occur in infancy, instances are recorded in older children which throw additional light on the etiology. In five cases of typical scurvy in children after infancy, observed by Dr. Barlow, the cause was traced to a curious morbid antipathy to vegetables and to meat.

In accordance with the fact that the majority of cases of scurvy occur in children fed upon patent foods and peptonised and other forms of prepared milk, comes out another curious fact; namely, that the disease is met with chiefly amongst the children of the better classes. Although the children of the poor are by no means exempt, the disease is much less common amongst them than amongst the children of the well-to-do.

Of the thirty-seven cases under my immediate observation during the last ten years, thirty-one occurred in private patients, and only six in hospital patients; and the experience of others is in accord with this statement. The unequal incidence is partly explained by the consideration that the artificial foods which are without antiscorbutic properties are chiefly used by the well-to-do. They are too expensive for the poor. The poor, however, use largely condensed milk and farinaceous materials, such as corn-flour and other farinaceous preparations; the first is feeble in antiscorbutic power, the latter destitute of it altogether. The reason why scurvy does not follow more frequently on diet of this kind is to be sought in the fact that the children of the poor begin to share the food of their parents at an earlier age than the children of the rich; thus they get a more mixed diet, of which potatoes, one of the most powerful of all antiscorbutics, usually form a chief part. The disease would appear to be growing more prevalent, in response, no doubt, to the more extended use of the dried and peptonised food preparations which now prevails. The experience of the American physicians is to a like effect. It is interesting to observe, in respect of the relation of scurvy to rickets, that this prevalence of scurvy amongst the rich, as compared with the poor, is the exact converse of the position of rickets in this respect; for rickets is most prevalent and most severe amongst the poor. The children of the poor grow rickety, the children of the rich scorbutic. The coexistence of rickets in the majority of cases of infantile scurvy is due to the fact that most of the foods—as notably the farinaceous and dried milk foods—are ricket-producing foods also, deficient in fat and proteid and phosphates of animal origin, as well as wanting in the fresh antiscorbutic element. It is possible also that the physiological activity of periosteal bone growth in infancy, and its vascularity, may be another factor in the meeting of scurvy and rickets.

Diagnosis.—The recognition of a case of infantile scurvy is not difficult when the typical signs of periosteal tenderness and swelling, and spongy gums, are present. When the latter sign is wanting, as may be the case in very young subjects in whom the teeth are not yet pushing, and the periosteal affection not pronounced, the condition is apt to be overlooked, or regarded as one of rickets, of rheumatism, or of simple anæmia and debility. Even in severe cases, the tenderness and swelling of the limbs leads not seldom to a mistaken diagnosis of rheumatism, from which, however, scurvy may be distinguished by the facts that the joints are free, and the part affected is the shaft of the bone above it, in addition to the other symptoms of scurvy present. Another common error has its origin in the motionless state of the lower limbs, which the child dreads to move on account of the pain; this inhibition of movement is frequently mistaken for paralysis, so that in many cases infantile scurvy is diagnosed as infantile paralysis. In other instances, again, in which the tenderness and dread of movement attract attention, the condition is regarded as one of tuberculous affection of the hip and knee joint. In another group of cases, in which hæmaturia or albuminuria are the symptoms first observed, the affection is regarded as a form of Bright's disease.

Even in cases where the gums are fungous, swollen, and bleeding, this local symptom has been regarded as the sole ailment, and the case judged to be a severe form of stomatitis. Similarly cases of proptosis due to scorbutic hæmorrhage may be regarded as cases of orbital tumour.

In all these conditions a full examination will seldom fail to reveal the true nature of the affection. Even if there be no sponginess of gums, the periosteal swelling, the exquisite tenderness of the limbs, the extreme dread of movement, and the earthy pallor and possibly hæmorrhages under the skin or elsewhere, added to the diet history, are sufficiently distinctive. If the gums be affected also, the evidence is complete, and the diagnosis may be quickly and finally confirmed by the test of antiscorbutic treatment.

All doubtful cases, especially those of rickets in which there is some limb tenderness, should be tested in this way.

The prognosis of infantile scurvy is almost uniformly favourable. If the disease is duly treated before extreme symptoms have arisen, recovery is rapid and certain. Before the nature of the disease was generally recognised the rate of mortality ran high. In the first series of thirty-one cases collected by Dr. Barlow seven proved fatal, or upwards of 21 per cent. Since that time, however, the death-rate has fallen remarkably. Out of thirty-nine cases under my immediate observation three only have ended fatally. Of these patients one was in a condition of extreme debility; the child took food with difficulty, and died from hæmorrhage into the lung three days after admission to hospital. In the second case the condition was also far advanced; the child was fed with difficulty, and died shortly after admission. In the third case the disease was not diagnosed at first, and the patient was treated surgically for a supposed abscess of the femur. It is to be noted that all the deaths occurred in hospital patients of the poorer class, in whom the disease had reached an extreme degree, under unfavourable conditions of life, before they came under treatment. In two, moreover, the difficulty of treatment was greatly enhanced by the fact that the curative agent could only be taken sparingly, owing to the extreme prostration of the patient; and death took place under these circumstances before sufficient time had elapsed for the amelioration of the disease. In the third case, in addition, the nature of the disease not being recognised, special antiscorbutic diet was not administered.

Danger to life exists, however, in all cases, both from hæmorrhages and from syncope, until the condition is controlled. In no instance within my knowledge has a fatal issue occurred after a week of antiscorbutic diet.

Treatment.—*Prevention.*—Since the disease arises from the persistent use of farinaceous dry foods or prepared foods containing no living or fresh element, or an insufficient amount of it, the obvious means of prevention lies in the avoidance of such foods, and in the use of some aliment of which fresh milk or other fresh material forms at least the chief part. Experience proves further that milk cannot with safety be submitted for any great length of time to predigestion, to prolonged heating at high temperatures, or to the process adopted in preparing preserved "humanised" milk. But to raise the milk to the boiling-point for a few moments, in what is called "scalding," does not appear to impair its nutritive value or its antiscorbutic power materially.

If therefore in any case the milk of the mother or of a wet nurse cannot be obtained, and diluted fresh cows' milk cannot be digested, the food in use should be replaced gradually by fresh milk, or some equivalent of fresh living material should be added. If, that is, it should be found absolutely necessary, on account of digestive troubles or other urgent reason, to place a young infant upon peptonised milk, humanised milk, or pancreatised food, or on any patent dried food, this should be regarded as a temporary expedient, and should not be continued for more than a few weeks. The prolonged use of such foods is a fruitful source of impaired nutrition in many ways, and especially of scurvy; it should be replaced by fresh milk by a system of gradual substitution. This can generally be effected by mixing fresh milk with the food used, in small quantity at first, and then slowly increasing it. Should the child be unable to digest a sufficient amount of the fresh milk in the course of a week or two, the lack of fresh elements may be supplied, and disaster averted, by the addition of raw meat juice, or by a small quantity of beef or chicken tea in which potatoes and carrots have been boiled and strained out. Similar precautions should be adopted in the case of older children when from illness or other cause they are placed upon a diet of peptonised or sterilised milk, or predigested food in any form, or upon any dried food to which fresh milk has not been added.

Cure.—The treatment of infantile scurvy consists almost entirely in the administration of fresh foods which possess the antiscorbutic virtue in high degree. The child should, if possible, be placed on fresh milk, which may be raised to the boiling-point, but not sterilised or "humanised." Fresh milk, however, as pointed out previously, possesses only moderate antiscorbutic properties, and is insufficient alone to effect the rapid cure of scurvy; just as it is insufficient to prevent it if given in small quantities.

For effective antiscorbutics we must have recourse to vegetable juices. Fresh green vegetables, more particularly the cruciferæ so efficacious in the scurvy of adults, are not available in the case of young infants of from six to eighteen months old, the period during which the disease usually appears. A most efficient substitute is, however, available in potato, which Dr. Baly, in his experience during the epidemics of scurvy at Millbank Penitentiary, proved to possess such remarkable antiscorbutic power. Even young children can usually take potato, properly prepared and administered, without digestive disturbance. It should be well steamed and reduced to light floury powder by rubbing through a fine sieve. This should be well beaten up with boiled milk until it is of the consistence of thin cream, and should be added to the regular food, beginning with a small teaspoonful to each bottle. The quantity may be gradually increased to a dessertspoonful, or even a tablespoonful in the case of children above a year old, if it agrees. Another effective plan, although less rapid than the preceding, is to administer the vegetable juices through the medium of beef tea or chicken tea, in which potatoes and carrots have been boiled and subsequently strained out. A small cup of this may be given once or twice a day.

The fresh element in diet should be further strengthened by the addition of the juice of raw meat, which possesses antiscorbutic power, although, like milk, not in high degree; and similarly it is unequal alone to effect the rapid cure of scurvy, or to prevent it when a small quantity is the sole addition to an otherwise scurvy diet. This comparative feebleness of raw meat juice and milk in antiscorbutic power have sometimes led to erroneous conclusions as to the nature of the disease, when it arises where milk or raw meat juice has formed a small portion of the diet, or has failed quickly to relieve it. Milk and raw meat juice are, in fact, only efficient when given in large quantity, and even then are much less active than vegetable juices. Raw meat juice has, however, a special value in these cases from its hæmic virtue. It contains iron in the most assimilable form in its hæmoglobin, and is the most powerful of all remedies for the anæmia constantly present and often extreme. The juice should be prepared by macerating the finely-minced pulp of raw beef in an equal quantity of cold water for half an hour, and then expressing all the liquid through fine muslin by twisting it. The straining is necessary to avoid danger of tapeworm by removing possible hydatids. It should be freshly made at the time of using, for it quickly undergoes decomposition, and, if kept, acquires poisonous properties.

Grape juice, orange juice, lemon juice, baked apples, are useful adjuncts, especially in the case of children above a year old. When potato pulp and raw meat juice are given and well borne, the result is immediate and almost magical. If the gums are spongy and swollen, all sign of this disappears in the course of a few days, the swelling of limbs goes down, tenderness subsides. In the course of a week or ten days the child no longer dreads handling or moving, and in a fortnight or three weeks is practically well—in striking contrast to the slow progress of simple rickets under similar dietetic treatment. In addition to antiscorbutic diet, fresh air and sunlight, as in the case of adult scurvy, are useful aids, although diet alone is certainly and rapidly curative. Little local treatment is required beyond wrapping the limbs affected in cotton wool, keeping the child absolutely at rest on a soft pillow, and preventing the movement of the limbs, which causes pain, and therefore wear and tear. The tenderness may be relieved, especially if the limbs are hot and uncomfortable from recent periosteal or muscular extravasation, by the application of warm compresses. As a rule, however, no local applications are required, and such measures as massage or stimulating applications are actively injurious.

Drugs are not required; diet is all-sufficient. Depressing remedies, such as iodide of potassium, often given with the mistaken view of aiding the absorption of the effused material of the subperiosteal swelling, are distinctly harmful; and iodide of iron is little less objectionable. Like all the iodides it is depressant, and if pushed far enough it eventually produces in children a cachectic purpuric condition.

Cod-liver oil and steel wine are useful in the later stages for any underlying rickets which may exist. In the active stage of scurvy they are better omitted, as they are apt to interfere with the ample ingestion of fresh food. In these cases raw meat juice is better than any iron preparation of the Pharmacopœia, and the cream of fresh milk is more potent than cod-liver oil.

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HÆMOGLOBINURIA

Synonyms.—*Paroxysmal hæmatinuria; Intermittent hæmatinuria; Intermittent hæmoglobinuria; Paroxysmal methæmoglobinuria.*

Definition.—Hæmoglobinuria is the name given to a disorder of which the most prominent symptom is the occurrence in the urine of hæmoglobin or methæmoglobin resulting from the destruction of red blood corpuscles in the general circulation. Various other names, as above, have been employed for the same pathological process.

Causation.—Destruction of the blood corpuscles, giving rise to the appearance of blood pigment in the urine, occurs under various circumstances:—

(i.) Exposure to extremes of temperature; as in cases of sun-stroke, severe burns, or frost-bite.

(ii.) The absorption of certain poisons into the blood through the alimentary or respiratory systems, or through the skin. Among these substances are sulphuric, nitric, hydrochloric, pyrogallic, and oxalic acids; arseniuretted and phosphoretted hydrogen, phenol, naphthol, quinine, nitro-benzine, poisonous fungi, and, perhaps more important than all of these, chlorate of potash.

(iii.) Transfusion, especially when the blood of an animal of one species is conveyed into the circulation of an animal of another species. In such cases the red corpuscles of the transfused blood are broken up during their passage through the blood-vessels of the animal into the circulation of which they are received.

(iv.) The disease occasionally occurs also as a complication of certain specific infectious disorders, such as scarlet and typhoid fever; as a result of certain blood diseases such as septicæmia, pyæmia, purpura, and scurvy; and of some unknown pathological condition of the blood-forming organs.

(v.) It occurs, again, as a primary disease, the so-called paroxysmal hæmoglobinuria, which is generally believed to be due to a previous invasion of the system by syphilis, malaria, or gout.

Raynaud's disease.—Hæmoglobinuria is not infrequently found in association with Raynaud's disease; even in comparatively mild cases deadness of the patient's fingers or toes and occasional "mottling" of the skin have been observed from time to time. On the other hand, many cases of Raynaud's disease have been recorded in which at no period of the affection could blood pigment or albumin be detected in the urine. Notwithstanding their frequent association, it may be doubted whether there be any relation between them other than that the paroxysms of both are apt to be induced by the same cause; namely, exposure to cold. In the one case the nerves and walls of the blood-vessels suffer, in the other the blood and possibly the blood-producing organs.

Condition of the urine.—This malady is to be carefully distinguished from hæmaturia, in which blood, as such, is found in more or less intimate admixture with the urine. In hæmaturia, according to the amount of blood present, the urine will be smoky, or even quite opaque; and under the microscope, blood corpuscles will be found in considerable numbers, particularly in the sediment which is deposited on standing. If the quantity of blood be large, coagulation will take place, giving rise to the formation of a definite clot in the urine. In hæmoglobinuria, on the other hand, the urine is generally clear when first passed, although on standing it may become more or less turbid. Its colour ranges from a light pink to a deep scarlet, brown, or black colour, according to the amount and state of the pigment present. In severe cases the colour may be as dark as that of porter. Its reaction is for the most part strongly acid, and on standing it deposits a thick precipitate of lithates, with which a quantity of hæmoglobin in an amorphous form is usually mixed; occasionally a very few blood corpuscles may be detected. The presence of blood, however, forms no integral part of the disease; it is merely the result of slight congestion of the kidneys, due to the irritation of the tubules by the passage of the disintegrated hæmoglobin. Occasionally the precipitated blood pigment is present in the urinary sediment in the form of minute yellowish, rounded masses which may be aggregated into the form of chains or bunches. Definite casts of the renal tubules composed of this material are sometimes found, but in all probability true hyaline casts do not occur. On rare occasions the hæmoglobin (or hæmatin) has been present in the crystallised form, and the occurrence of crystals of oxalate of lime has also been noticed.

Should there be any doubt as to the true nature of the extraneous colouring matter in the urine the various tests for the presence of blood or its derivatives may be applied. Examinations by means of the microscope will determine the presence or absence of the red blood corpuscles; although it is well to remember that their shape and consistence may vary considerably with the reaction and specific gravity of the urine. If the colouring be not very intense, special search should be made for the corpuscles in the sediment deposited on standing. If none be found, the guaiacum test for blood pigment may be applied; but the test is somewhat fallacious, as the same colour reaction may be obtained in the presence of pus or mucus. The only really trustworthy evidence will be afforded by the spectroscope, by which not only can the presence or absence of blood pigment be determined, but also the actual form in which it is present. In making the spectroscopic examination care must be taken to use only such a strength of solution that the light can easily pass through it. If the colouring matter is blood pigment, the spectrum of hæmoglobin (Fig. 1, Nos. 1 and 2) or of methæmoglobin (No. 3) may be seen. The latter is almost identical with the spectrum of acid hæmatin (No. 4), from which, however, it can be distinguished by addition of some reducing agent to the urine. If the spectrum be due to methæmoglobin, the pigment will become converted into reduced hæmoglobin, which gives a single wide absorption band in a position intermediate between the two bands due to oxyhæmoglobin. If, on the other hand, the original spectrum were due to the presence of acid hæmatin (No. 4), a single intense band of reduced alkaline hæmatin (No. 6) will be observed on the violet side of the sodium line between D and E, and, in strong solutions, a much fainter band still farther towards the violet end of the spectrum.

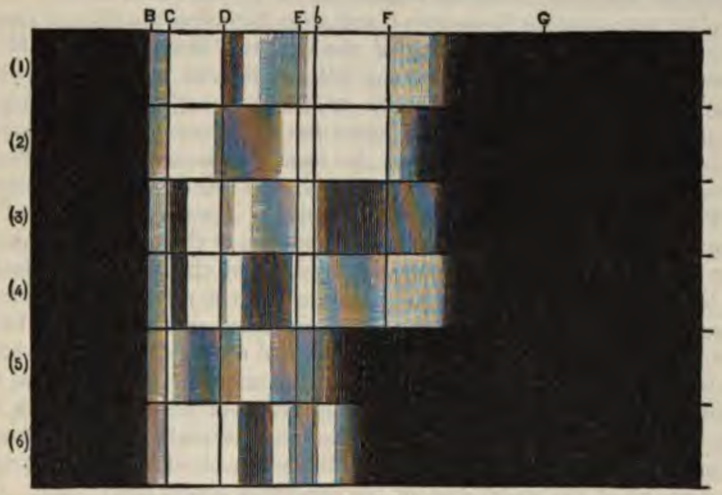

FIG. 32.—Spectra of hæmoglobin and its derivatives. (1) Oxyhæmoglobin; (2) reduced hæmoglobin; (3) methæmoglobin, (4) acid hæmatin; (5) alkaline hæmatin; (6) reduced alkaline hæmatin.

Owing to the presence of hæmoglobin, or its derivatives, the urine will generally contain a more or less considerable quantity of proteid which, although when the urine is heated it gives rise to a coagulum (reddish brown in colour, owing to the pigment entangled with it), is probably not serum albumin, as in the ordinary forms of albuminuria, but consists for the most part of globulin, as was first pointed out by Sir William Gull.

The coagulum formed by heating the urine is also said to differ from that obtained in like manner in the urine in Bright's disease, in that it usually floats on the surface of the fluid instead of sinking immediately to the bottom. The proteid, the presence of which may be demonstrated in this manner, or by the other general tests for bodies of this nature, is derived from the splitting up of the hæmoglobin present.

This point may be demonstrated by saturating the specimens of urine with magnesium sulphate, by which reagent globulin is precipitated, but not serum albumin. The specimens should be left some time to ensure complete precipitation if possible; and after filtration the filtrate may be treated with a further quantity of the salt, and filtered again. If any serum albumin be present in the filtrate it will be precipitated by slightly acidifying with dilute acetic acid, and then gradually raising the temperature to about 80° C. In some few instances a certain amount of nephritis may be associated with the hæmoglobinuria, in which case, of course, serum albumin would be present in the urine as well as globulin.

Pathology.—Hæmoglobinuria is probably always the result of the removal of hæmoglobin from the red corpuscles in the general circulation. That this is so may be demonstrated by cupping the patient and leaving the blood, in a small cylindrical glass vessel, in a refrigerator for a considerable length of time. The red corpuscles gradually sink to the bottom, when, if the blood has been obtained during an attack of hæmoglobinuria, the serum will be tinged with a more or less deep red tint. A simpler method of testing for the presence of free hæmoglobin is to remove serum from a blister and examine it with the spectroscope. Normally, the only absorption band visible when blood serum is examined is a dim one at the violet end of the spectrum, about the F. line. This is indicative of the presence of lutein, to which the colour of the serum is said to be due. If, on the other hand, the serum has been obtained from a blister at the beginning of an attack of hæmoglobinuria, the characteristic absorption bands of hæmoglobin will present themselves, their intensity being proportional to the thickness of the layer of serum examined. It was formerly taught that hæmoglobinuria is primarily a disease of the kidneys, but no evidence has been afforded to justify this opinion; in fatal cases of the disease no definite alteration in the structure of the kidneys has been demonstrated.

TOXIC HÆMOGLOBINURIA.—One of the most important, because the most dangerous, forms of the diseases included under the general name of hæmoglobinuria is that variety which occurs as a result of poison.

Mention has already been made (p. 621) of the different ways in which hæmoglobinuric intoxication has been induced; of the mode of its causation but little is known; especially as, with regard to the majority of these toxic agents, the recorded instances of their action are but few in number. It is a remarkable fact that the employment of chlorate of potash has been followed in a considerable number of cases by a severe attack of hæmoglobinuria, which has often resulted in death. Thus it was in no less than 23 out of 27 cases collected by Hofmeier. This untoward result, indeed, has not infrequently ensued on the ingestion of unusually large doses of this drug, whether administered intentionally, as a therapeutic agent or with suicidal intent, or in a strong solution, intended for use as a gargle, but unfortunately swallowed by mistake. The urine passed in these cases of poisoning, in that it contains a large amount of the dark granular débris of the broken-down corpuscles, resembles that usually seen in other forms of hæmoglobinuria. The amount of urea present has also often been enormously increased. The blood pigment present is invariably in the form of methæmoglobin, which has also been detected in the circulating blood—a point in which this form of hæmoglobinuria differs from the paroxysmal variety. Tomaselli has also recently put on record a number of cases in which symptoms resembling paroxysmal hæmoglobinuria have followed the administration of quinine to certain patients who were the subject of chronic malaria; this is a matter of no little importance, as this drug is often employed in the treatment of hæmoglobinuria, especially when attributed to malaria. Tomaselli believes that the method of administration, and the quantity of the drug employed, have little bearing on this curious idiosyncrasy, which appears indeed to be more or less transmissible, since several members of the same family showed the same intolerance of the drug.

Symptoms.—In Tomaselli's cases, half an hour to a couple of hours after the quinine was given, the patients were suddenly seized with nausea and shivering, accompanied by a considerable rise of temperature. Complaint was also usually made of a feeling of weight in the loins followed by an imperative need to void urine, which when passed was found to be "sanguineous." Not infrequently vomiting, diarrhœa, and jaundice also ensued.

In chlorate of potash poisoning the chief symptoms are very similar, the patient being seized with rigors followed by vomiting and diarrhœa. Eventually he becomes collapsed and comatose, and dies after a variable interval. The fatal dose of this drug has been set down in the adult at from three to four drachms, or less, in the twenty-four hours; in the case of young children about half this quantity has been known to cause death.

Morbid anatomy.—In cases of toxic hæmoglobinuria the kidneys are generally found to be of a more or less uniformly dark brown colour; under the microscope the renal tubules are seen to be plugged with a brownish granular material which is often found also in the Malpighian capsules. The colour of this material, and also of the kidneys generally, is due to the conversion of the pigment into methæmoglobin. No constant change has been noted in any other organ.

Treatment.—No drug is known to exert a direct influence on hæmoglobinuria. Treatment must therefore be directed to removal of the cause, if this be possible. The more disturbing symptoms must be allayed, and the patient placed under such favourable conditions as warmth and rest in bed.

INFANTILE HÆMOGLOBINURIA.—Occasionally this disorder occurs among infants; in some cases it seems to alternate with true hæmaturia. Usually no general symptoms are present, and the child does not show any signs of pain; the only indication of anything wrong is the appearance, at more or less irregular intervals, of blood or blood pigment in the urine. Even this indication is wont to disappear on admission to hospital, where equable temperature, regular and proper feeding, and attention to the digestion effect the patient's cure, at any rate for the time being. This affection seems to show that an occasional extra-physiological destruction of corpuscles may be a result of improper feeding, clothing, and the like carelessness, on the part of ignorant or inattentive parents. The proper method of treatment in such cases is obviously hygienic.

Now and again, however, more serious outbreaks occur, such as that put on record by Winckel, which occurred in the wards of a lying-in hospital at Dresden in the spring of 1879.

Here, during a period of about six weeks, twenty-four newly-born infants were attacked with a form of hæmoglobinuria; of these no less than twenty-three died. In all these cases the symptoms were practically identical, and were very similar to those met with in the toxic form of the disease. Thus the children within a few days of birth showed signs of collapse, and the skin acquired a yellowish tinge. This was followed by a distinct rise of temperature, and by increase in the pulse and respiration rate. The urine was somewhat scanty and brown, the contained pigment probably consisting, in part at any rate, of methæmoglobin. Death ensued in about thirty-six hours from the beginning of the illness. The necropsies revealed considerable enlargement of the mesenteric lymph-glands and of the spleen—the latter organ being somewhat tougher than usual and of a browner colour. The kidneys also were of a brown colour, and the renal tubules were plugged with masses of hæmoglobin.

The onset of this affection appeared to be due to a more or less complete disintegration of the red corpuscles of the circulating blood; but, though investigation was diligent, no sufficient cause for the outbreak could be discovered. It is not improbable, however, that some bacterial infection played a part in the matter. The general symptoms and the post-mortem appearances all pointed to a toxic cause; and other facts, which appear to support such a contention, are that in each instance a period (of incubation ?), of about equal length in all, elapsed between the birth of the infant and the invasion of the system by the disease; that a large number of children were affected within a comparatively short period, thus giving an indication of possible infection; and that the disease was as sudden in its disappearance as in its first onset.

PAROXYSMAL HÆMOGLOBINURIA.—**Definition.**—A disease, not dependent on any known anatomical lesions, in the course of which the patient is attacked, at more or less irregular intervals, by severe rigors, followed, after a longer or shorter period, by a discharge of urine ranging in colour from a pinkish hue to a bright scarlet, or even black-brown; such colour being due to the presence of a quantity of blood pigment in the form of hæmoglobin, or of one of its derivatives. The disease was first described by Dr. George Harley under the name of intermittent hæmaturia, and shortly afterwards by Sir William Gull as intermittent hæmatinuria. To Dr. Pavy we owe the more accurate name Paroxysmal Hæmoglobinuria, now generally used.

Causation.—In some cases attacks of this disease may occur even in the height of summer; nevertheless, the most obvious immediate cause is exposure to cold. Such exposure in the first instance was often extreme; but where the tendency already exists, a comparatively slight chill is sufficient to determine an attack. As a general rule the patient is free from attacks during the warm weather; but with the return of winter the affection reappears, although even then the malady may remain in abeyance as long as confinement in an equable temperature at home or in hospital is observed. The liability to attack may persist for years without much apparent danger to life; although a severe series of paroxysms may seriously depress the vitality of the sufferer for the time. Exhaustion of any kind, whether mental or bodily, over-work, excesses of the table or of the sexual functions, or again, want of proper nourishment of the body, whether resulting from dyspepsia or from the actual deprivation of food, undoubtedly dispose to attack.

The disease is almost entirely confined to men, usually between the ages of fifteen and fifty or sixty years. Attacks, however, have been recorded in women; and in certain instances the disease has been known to occur, in its most typical form, in children even of quite tender years. The disease has probably some affinity to syphilis, whether acquired or congenital; a definite specific history has been forthcoming in all the cases that have come under my own observation.

In many cases there has been a history of malaria also; although, as this form of hæmoglobinuria is apt to occur in malarious countries, the connection between the two diseases may have been assumed to be more definite than it really is. Gout and rheumatism have also been placed among the remoter causes of the disease.

Symptoms.—Generally after definite exposure to cold the patient is attacked, at a longer or shorter interval, with chilliness of the extremities, often attended with dead fingers or toes, shivering or actual rigors, pallor and roughness of the skin, general sensation of cold, and often severe headache. He may complain of pain or difficulty in swallowing; although there is usually no loss of appetite, and no evidence of disease either in the thoracic or in the abdominal viscera. In this early stage the temperature of the body is usually lowered by as much as two or three degrees. Within from half an hour to three hours a quantity of urine is passed which is of a somewhat high specific gravity, of a red, brown, or black colour, clear, acid, and containing excess of urea and abundant albumin. Occasionally the urine is turbid when passed, and in any case on standing it deposits abundant sediment, composed for the most part of a brownish granular matter. Occasionally some of the blood-pigment is deposited in a crystallised form also. During this period the patient, if he is in the house, usually crouches over the fire, and feels sick and giddy, even if he do not actually vomit. A reactionary rise of temperature now ensues, which may reach as high as 103° F. The whole attack usually lasts for about five hours, after which time the urine gradually loses its specific characters, and the patient very shortly seems to be restored to much the same condition as that in which he was before the onset of the paroxysm.

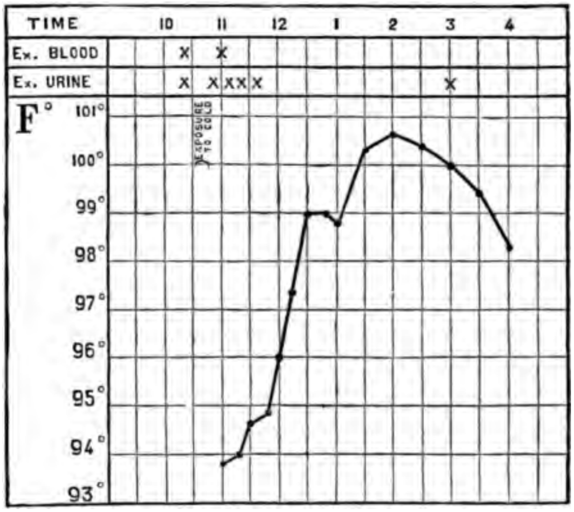

CHART 6.—Chart showing range of temperature during a typical attack of paroxysmal hæmoglobinuria.

Similar attacks will recur as frequently as the patient is exposed to the causes. They may return with great regularity, and sometimes two or three attacks may occur during a single day; but they are not characterised by the definite periodicity which is seen so notably in ague. If, on the other hand, the patient's surroundings be favourable, attacks may be postponed for an almost indefinite period. In a patient subject to the disease, the skin, particularly of the face, is generally of a somewhat sallow colour, which is apt to become intensified after an attack; so much so indeed, on occasion, as almost to simulate jaundice. Considerable anæmia may also be noticeable, the patient sometimes remaining weak and languid in the intervals of attack; and it may be possible to observe capillary pulsation on the lips, such as is often obtainable in other cases of extreme anæmia, and generally in aortic regurgitation.

Pathology.—One of the most remarkable features of this disease is the enormous and often extremely rapid destruction of the red blood corpuscles, such destruction depending apparently on the direct influence of cold. This is well shown in the series of experiments carried out by Dr. Bristowe and myself, in which the blood was examined, by means of the hæmocytometer, immediately after exposure to cold, and before any blood pigment had been passed by the urine. The results of a number of cases on which such examinations were made were so far identical that a large decrease in the number of corpuscles was noticed, varying from 129,000 to 824,000 per cubic millimetre. The injurious action of cold on the blood corpuscles was further shown by the loss of consistence and the variability of size and shape of those which survived; by the presence of granular masses of hæmatin in the plasma, and by the tinting of the plasma with the escaped hæmoglobin of the corpuscles. In the case of three children who came under my observation at the Great Ormond Street Hospital, the direct influence of cold was specially obvious, and it may be mentioned that two adult patients, who remained for a considerable time in St. Thomas's Hospital, had no attack while there, save those which were brought on by occasional exposure to cold.

The extreme rapidity with which the destruction of the corpuscles is effected was shown in some cases by the examination of the blood immediately after the patient had been exposed to cold, and before any of the characteristic symptoms of an attack had been observed; but there is also ground for believing that the destruction of the corpuscles goes on at a diminished rate for some little time after such exposure. The removal of pigment from the corpuscles does not usually affect the urine for half an hour or more after the beginning of the exposure.

A proteid is excreted with the pigment which may readily be shown to be globulin and not serum albumin.

Dr. MacMunn has put the statement on record that the pigment invariably consists of methæmoglobin; but if the urine be drawn off from the bladder by means of a catheter at frequent intervals from the onset of the attack, it can be shown then to consist of oxyhæmoglobin.

Dr. Druitt, who himself was the subject of this disease, states that his urine was of a bright scarlet colour on those occasions when, suffering great pain from the irritability of the bladder, he was obliged to void urine about every half-hour. On the other hand, if the pigment be allowed to remain in the bladder for some considerable time in contact with the acid urine, it may finally become converted into methæmoglobin, or even into acid hæmatin. Experiments show, however, that it invariably passes through a preliminary stage of methæmoglobin before conversion into acid hæmatin. Some difficulty in distinguishing these pigments has evidently been experienced in the past, as the spectrum of methæmoglobin and that of acid hæmatin are practically identical, each showing four absorption bands (Hoppe-Seyler); although in most text-books only three bands are given for methæmoglobin. In specimens of urine obtained from persons suffering from paroxysmal hæmoglobinuria the four absorption bands are generally well marked. This fact may have led to the conclusion that the pigment voided is acid hæmatin, even in cases in which methæmoglobin was present alone. In point of fact, however, the degree of change appears to depend solely on the length of delay in the bladder (or perhaps, in part, in the renal tubules); so that, according to the time which the urine has remained there, oxyhæmoglobin, methæmoglobin, or even acid hæmatin is obtained.

Hæmoglobin, the colouring matter of the red corpuscles, is known to be capable of existing in the three forms of oxyhæmoglobin, reduced hæmoglobin, and methæmoglobin. These differ from one another, not only in the amount of oxygen in combination but also in the colour of their solutions, and in their absorption spectra. The reaction of the first two of these modifications is alkaline. By splitting up hæmoglobin hæmatin is produced which also is capable of existing in three forms; one of these, which is very stable, and has an acid reaction, is known as acid hæmatin or hæmatin in an acid solution; the two others possess an alkaline reaction, but differ in the amount of oxygen in combination. Methæmoglobin, on the other hand, has an acid reaction, and its spectrum is almost, though not quite, identical with that of acid hæmatin. It is readily distinguished from this body, however, as, when treated with a reducing agent such as ammonium sulphide, or, better still, sodium hyposulphite, it is changed into reduced hæmoglobin, while acid hæmatin under similar circumstances yields reduced alkaline hæmatin. It is evident that methæmoglobin must be nearly related to hæmoglobin, although there has been some diversity of opinion on the subject. Recently, however, it has been proved conclusively that it contains precisely the same amount of oxygen as oxyhæmoglobin, from which, therefore, it differs only in its closer union with its oxygen, and in its acid reaction.

It is noteworthy that not only, as has been stated, is the destruction of blood corpuscles rapid and enormous, but that the restoration of blood corpuscles is also remarkably rapid; experiment has shown that in the course of from four to six days after a severe attack their number will have risen almost to the amount recorded in the previous interval of health. It appears highly probable, indeed, that paroxysmal hæmoglobinuria is but an exaggeration of a physiological phenomenon. The red corpuscles of the blood are constantly undergoing destruction, the products of this destruction are used up in the system, and in health they do not appear in the urine either in the form of hæmoglobin or of albumin. If, however, the destruction oversteps the normal limit, the system is unable to make away entirely with the products of such destruction, and albumin appears in the urine. If the destruction be much above the limit, even for a very short time, then oxyhæmoglobin will appear; or, if the pigment be retained in the tubules of the kidney or in the bladder for any length of time, methæmoglobin.

Sir George Johnson, Dr. Mahomet, Dr. Ralfe, and others have called attention to the fact that temporary albuminuria may follow cold bathing, or any other form of exposure to cold, in persons apparently healthy; and a case has been recorded by myself, in which an attack of hæmoglobinuria followed a cold bath taken after exertion at tennis, in an athletic man, apparently in perfect health, who has never had any symptoms of renal disease either before or after this solitary attack. Dr. Ralfe also showed from his own personal experience that albuminuria is apt to occur in persons otherwise apparently healthy after exposure to cold, fatigue, or mental worry; and—excepting that there was no rise of temperature—with symptoms practically identical with those characteristic of hæmoglobinuria. Indeed, Ralfe expressly stated that in four of his cases the attacks of paroxysmal albuminuria occurred in persons who had been subject to hæmoglobinuria; and he considered that there is a definite relationship between the two diseases. It appears extremely probable that paroxysmal globulinuria (which name appears preferable to the more usual one of albuminuria, at any rate for those cases which are brought on by exposure to cold) is a latent form of paroxysmal hæmoglobinuria, being due, as in this latter disease but in less degree, to abnormal destruction of the red corpuscles in the blood.

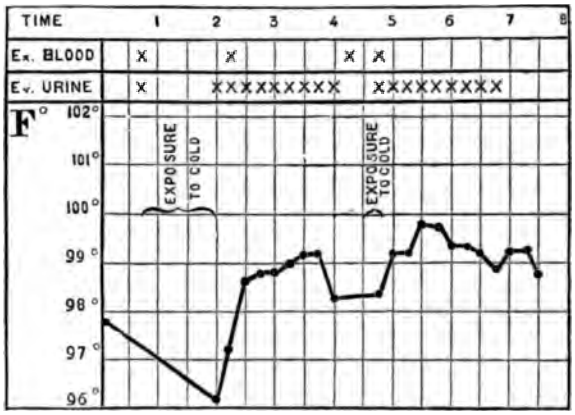

CHART 7.—Temperature curve in a case of paroxysmal hæmoglobinuria.

That a relationship between these two affections actually exists appears to be proved by the effect produced on persons subject to paroxysmal hæmoglobinuria by exposure to slight degrees of cold. Such exposure is often followed by a marked elevation of temperature, together with comparatively slight but unmistakable destruction of corpuscles; the evidence of which is the appearance of globulin (not albumin) in the urine. This relationship is well brought out in a series of observations made by myself in the case of an omnibus conductor, aged 41, who for two years previously had suffered during the winter months from occasional attacks of paroxysmal hæmoglobinuria. In the accompanying Chart 6 it will be seen that, as judged from the temperature curve, the first exposure to cold, which consisted in the taking of a short walk in the open air, resulted in less constitutional disturbance than did a second but shorter period of exposure later in the same day.

At intervals of time, designated by crosses marked on the chart, examinations of the urine were carried out, the results of which are shown in tabular form. During the first period covered by these observations no blood-pigment could be detected in the urine, although albumin (globulin) was found in easily recognisable amount.

Temperature.	Pulse.	Resp.	Urine.	
			Albumin.	Blood-colouring matter.
Before exposure 97·8°	...	...	None	No guaiacum reaction, and no absorption-bands
2 P.M. 96·2°	76	14	None	
2.15 P.M. 97·3°	82	18	None	
2.30 P.M. 98·6°	73	17	Trace	
2.45 P.M. 98·8°	85	13	Larger trace	
3 P.M. 98·8°	85	15	Larger trace	
3.15 P.M. 99°	80	14	One-tenth	
3.30 P.M. 99·2°	87	16	Fair trace	
3.45 P.M. 99·2°	82	15	Trace	
4 P.M. 98·4°	82	14	None	

Some time later, after the second period of exposure, the slightly higher range of temperature which ensued was accompanied by the additional presence of blood-pigment in the urine.

Temperature.	Pulse.	Resp.	Urine.	
			Albumin.	Blood-colouring matter.
4.45 P.M. 98·4°	84	17	None	None
5 P.M. 99·2°	87	17	Trace	Guaiacum reaction
5.15 P.M. 99·2°	76	16	Much	Hæmoglobin (spectroscope)
5.30 P.M. 99·8°	84	16	One-quarter	Do.
5.45 P.M. 99·7°	78	17	Less	Do.
6 P.M. 99·4°	73	15	Trace	Do.
6.15 P.M. 99·4°	72	16	Trace	Do.
6.30 P.M. 99·2°	74	16	Trace	Trace
6.45 P.M. 98·0°	74	16	None	None

These results accord to some extent with the experiences of Ponfick, who has shown that, whilst an injection of large quantities of hæmoglobin into the blood causes hæmoglobinuria, the injection of small quantities causes no such elimination. It seems a legitimate inference, therefore, that when the unwonted destruction of red corpuscles, the cause of which has been exposure to cold, is comparatively slight, the proteid moiety of the hæmoglobin alone appears temporarily in the urine; the colouring matter of the effete corpuscles, on the other hand, being used up in the system, probably by conversion into biliary and urinary pigments. When, however, the destruction is more extensive, hæmoglobin is discharged as such.

In the light of these arguments it is not necessary to assume that either of these affections is dependent upon disease of the kidneys, which appear to act merely as the organs for the elimination of the excess of effete products with which the blood is charged.

It must be admitted, however, that, although the destruction of red blood corpuscles is the most obvious feature of paroxysmal hæmoglobinuria, there is very strong evidence to show that there is some antecedent peculiarity of the blood corpuscles themselves which renders them unduly sensitive to the influence of cold, seeing that in the healthy man an equal degree of exposure is quite incompetent to bring about such a result. According to Murri, the cause is to be sought in the diseased condition of the blood-forming organs, which renders the corpuscles less resistant to cold. This is borne out by the fact that under the microscope the blood corpuscles do not run together into rouleaux in the normal manner, even when there has been no paroxysm for some time; while their consistence seems to be lessened, as, in preparations for the microscope, the slightest pressure on the cover-glass is often sufficient to make them assume all kinds of fantastic shapes. One cause for this chronically diseased condition of the blood-forming organs may probably be found in the effects of syphilis, as has already been suggested by Murri, Barlow, and others. As bearing on this point it is of interest to note that in all my own cases syphilis, either primary or congenital, had been present. It is hardly possible to look upon such an occurrence as a mere coincidence.

As far as our knowledge at present extends, paroxysmal hæmoglobinuria appears to depend on two main factors:—

1. A lessened power of resistance on the part of the blood corpuscles, due to some defect in the blood-producing organs.

2. A tendency on the part of the corpuscles to break down in the general circulation under the influence of cold, followed by the appearance in the urine of the products of such hæmolysis.

This tendency to disintegration of the corpuscles is apparently the result of an imperfect power of production in the blood-forming organs, caused in turn by the baneful influences of syphilis, or possibly of malaria, gout, or rheumatism.

Treatment.—Seeing that the paroxysms of this disease appear to be determined for the most part by exposure to cold, the obvious indication is to keep the patient as much as possible in a warm and equable temperature. This is to be done in severe weather by confinement to the house, and by protecting the body from the effects of possible draughts by means of warm clothing, which, especially as regards the under-garments, should be of wool. Meals should be regular and ample, but the food should be of an easily digestible kind. Alcoholic drinks, especially wine and spirits, unless much diluted, should be avoided, as they tend to produce a temporary dilatation of the superficial capillaries of the skin, which may bring about a slight lowering of the body temperature. Worry of mind or body should be avoided as much as possible, and the patient should keep early hours, so as to escape the imprisonment in heated rooms which appears almost inseparable from evening entertainments. But only by removal to a warmer climate than that of England can there be a reasonable prospect of curing the disease.

The attack will probably be relieved if the patient retire to bed immediately it threatens. He must be kept warm by every possible means, such as a sufficiency of bed-clothes, and hot bottles in the bed. Whether excessive thirst be present or not, a cup of hot soup will probably be found both pleasant and useful.

Probably but little good is to be obtained from treatment by drugs, although both quinine and arsenic have by some been vaunted almost as specifics for the disease. On the other hand, I must repeat that Tomaselli has shown that, in some instances, the administration of quinine has appeared to induce an attack of hæmoglobinuria. If quinine be employed it should be given in full doses, but no considerable benefit can be expected from it in severe cases.

As from the constant drain on the blood system a certain amount of anæmia is usually present, this symptom should be met by an administration of iron, either alone or combined with arsenic or digitalis. Seeing that syphilis is present in a considerable proportion of cases, iodide of potassium and the various forms of mercury should invariably be employed, when for this reason their use is indicated; in some instances, indeed, permanent cure has followed this method of treatment.

S. MONCKTON COPEMAN.

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S. M. C.

LEUCOCYTHÆMIA

LEUCOCYTHÆMIA, or Leuchæmia (Ger. Leukämie), may be briefly described as a disease in which there is great, and usually permanent, increase in the number of leucocytes in the blood, associated with greater or less anæmia, and with peculiar changes in the spleen, bone-marrow, lymphatic glands, or other organs, these being affected in various combinations. Further, the leucocytes are not only increased in number, but, taken as a whole, are altered in character also.

Introductory.—Though conditions which can now be identified as cases of leucocythæmia had been described before, the definition of the group of symptoms of which it consists dates from the independent and almost simultaneous publications of Hughes Bennett and Virchow on the subject. In October 1845 the former recorded a case which, from the microscopical characters of the blood, he described as one of "suppuration of the blood, with enlargement of the spleen and liver"; and a month later Virchow gave an account, under the title "white blood," of a similar condition in which he noted the association of splenic enlargement, epistaxis, and a peculiar richness of the blood in leucocytes. In both of these cases the important changes were observed after death. Afterwards Bennett gave the name *leucocythæmia* to the disease, whilst Virchow called it *leukaemia*. A short time after his first case Virchow observed another in which the leucocythæmic condition of the blood was associated with enlargement of the lymphatic glands, whilst the spleen was only slightly enlarged; and in subsequent papers he drew a distinction between a *lymphatic* form of the disease in which there is an admixture in the blood of leucocytes from the enlarged lymphatic glands—"lymphæmia," and a *splenic* form in which he believed the excess of leucocytes to be produced in the enlarged spleen—"splenæmia"; the cells in the blood being of smaller size in the former than in the latter variety. Neumann, in 1870, not long after his discovery of nucleated red corpuscles in the bone-marrow, found that this tissue is often profoundly altered in cases of leucocythæmia, and this change he regarded as primary. After much discussion of Neumann's views a *medullary* form was added. A considerable amount of confusion, however, arose from this classification of cases according to the organs affected, as, according to this nomenclature, most cases were found to be of a mixed kind.

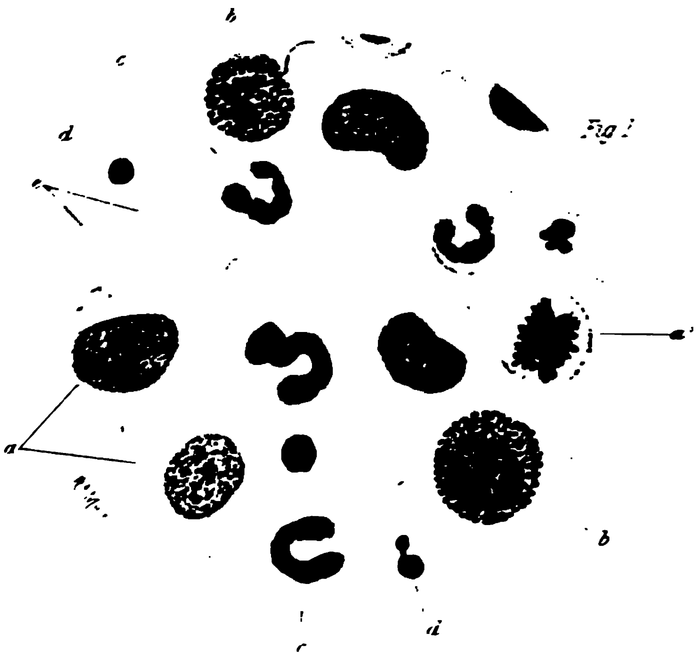
Renewed interest in the subject was aroused by the researches of Ehrlich and others on the characters of the cells in the blood and their reactions to various aniline stains; and much of the work in recent years has been along the same lines. The general result has been a tendency to take the characters of the leucocytes in the blood in leucocythæmia as the basis of distinction in different cases, and this method appears to me to be justifiable.

Within recent years special attention has also been directed to the more acute forms of the disease; and, in consequence, cases which formerly would have been overlooked have been identified as cases of leucocythæmia. Though the disease is, as a rule, very chronic, yet there are instances in which it runs its course in a few weeks after the first appearance of symptoms. In these the splenic enlargement is often slight, and the true nature of the condition is revealed by examination of the blood.

VARIETIES.—Taking as the distinctive feature the characters of the leucocytes found in excess in the blood, we find that there are two chief varieties.

A. In the one class, which includes most of the cases generally described as splenic leucocythæmia, there is almost invariably great splenic enlargement, whilst there are present in the blood numerous large uninucleated cells and certain other elements which are believed by many authors to originate in the bone-marrow. The latter point will be discussed later. The name *spleno-medullary* is often applied to this, the commoner, variety of the disease, and will be adopted in this article. It might be more correct to speak of it as medullary or myelogenic leucocythæmia, or myelocythæmia with splenic enlargement; but the origin of the disease in the marrow has not yet been proved. Some German writers have called this form "myelæmia." In it the lymphatic glands are not usually enlarged, but may be so, especially late in the disease. Though the great splenic enlargement is almost constant, a few cases have been recorded in which it was absent or slight; and these have been designated as cases of *pure medullary leucocythæmia*. Provided that the condition of the blood be the same, they may be classified with the others.

B. In the second variety the leucocytes in excess in the blood are chiefly of the small uninucleated class, that is, are lymphocytes. This will be spoken of as the *lymphatic* form, though the name *lymphocythæmia* might be adopted. In this form the lymphatic glands are usually enlarged, though occasionally the enlargement may be slight or even absent;



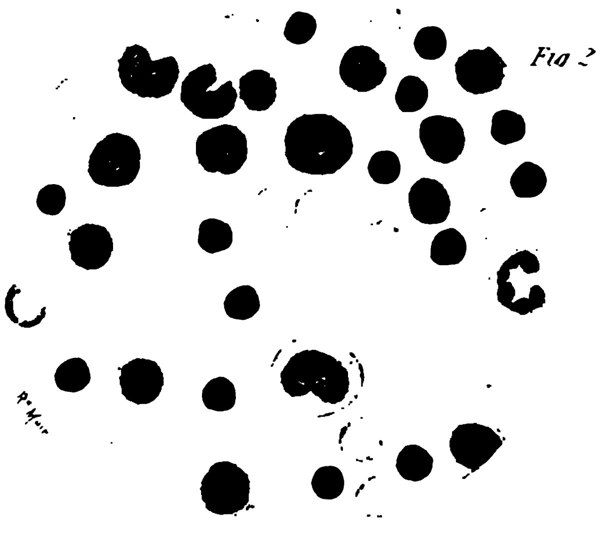


Fig. 1.—Spleno-medullary leucocythæmia. Film of blood, showing the chief forms of cells met with: (*a*) Large uninucleated cells, "marrow cells," one of which (*a'*) shows mitosis; (*b*) eosinophile cells; (*c*) ordinary leucocytes with multipartite nucleus; (*d*) nucleated red corpuscles, showing variety in form of nucleus; (*e*) ordinary red corpuscles. × 1000.

.—*Lymphatic leucocythæmia* or lymphocythæmia. Film of blood showing enormous increase *uninucleated leucocytes*, many of which are of very small size, with scarcely visible protoplasm. *000.*

splenic enlargement is the rule, though it is not generally so great as in the first form: other organs are not infrequently the seat of diffuse leucocytic infiltrations. It is not possible to infer the origin of the condition from the characters of the cells in excess, as the lymphocytes have such a wide distribution in the normal body; but that in many cases the disease may originate in the lymphatic glands.

If we attempt to classify cases of leucocythæmia according to the organs affected, we are met at once by the objection that usually more than one are affected at the same time; moreover, we do not find that the characters of the leucocytes in the blood vary with the organs affected. In a pronounced case of spleno-medullary leucocythæmia, for example, enlargement of the lymphatic glands may occur, but this enlargement is not attended by the appearance of lymphocytes in the blood; so also the spleen may be very much enlarged in the lymphatic form (lymphocythæmia), whilst the lymphocytes alone are increased in the blood. In the latter case the splenic enlargement is found to be due to distension of the pulp with lymphocytes, a change analogous to what may be found in the kidneys and other organs; the bone-marrow also may be the seat of lymphocytic infiltration. As the increase of leucocytes, both in the blood and in the various organs affected, is the essential feature of the disease, it is better to take differences in their characters as the principle of classification; especially as these differences are of a definite nature.

PATHOLOGICAL ANATOMY.—**The blood.**—The appearance of the blood may show little change on naked-eye examination, or, on the other hand, it may be strikingly altered. In cases where the number of leucocytes is very much increased, it is pale and slightly turbid in appearance, as if mixed with pus; and if, in addition, marked anæmia be present, it is thin and watery and may have a yellowish tint. It usually coagulates less readily than normal blood, especially when there is much anæmia.

On microscopic examination the change is generally obvious at once, the number of leucocytes being notably in excess. But, as will be shown hereafter, it must not be inferred that when the increase is apparently trifling the case is not one of leucocythæmia. Occasionally the number may fall very considerably in the course of the disease, or even towards death.

As the characters of the leucocytes have been taken as the means of classification, and as they differ considerably in the two forms, they will be described under these two heads.

A. *Spleno-medullary form* (*vide* Fig. 1 of Plate).—In the fresh blood a large proportion of the leucocytes are seen to be of greater size than those of normal blood; namely, 14-16 μ in diameter. Most of these are finely granular, whilst some contain coarse, highly refracting granules. The characters of the nuclei may be made visible by the addition of weak acetic acid, but they are best studied in films of blood prepared by Ehrlich's dry (or a corresponding) method (*vide* p. 413), and afterwards stained; for the study of the granules this is necessary. In such preparations a great variety of cells is found to be present, some of which are not normally present in the blood. The following are the chief forms:—

1. Large uninucleated corpuscles, which are often called marrow-cells or myelocytes ("cellules medullaires" of Cornil) from their supposed origin. These are the largest cells present, and form a considerable proportion (sometimes more than half) of the total number of leucocytes (Fig. 1, *a*). The single nucleus is of large size, and is round, oval, or indented at one side so as to have more or less a horse-shoe shape. It stains rather faintly, being weak in chromatin, which forms a loose network with granular thickenings in its interior. The protoplasm is finely granular, and stains diffusely and faintly with a nuclear stain, such as methylene blue. The minute granules in the protoplasm have, according to Ehrlich, a "neutrophile" reaction—staining with a mixture of a basic and an acid aniline stain; according to others, a weak "oxyphile" reaction. Some of these cells may be found undergoing mitotic division (Fig. 1, *a′*), though mitotic figures, as a rule, are not many, and may be sought for in vain. When examined on a warm stage nearly all these corpuscles are quite devoid of amœboid movement, only some of the smaller forms showing traces of it.

2. Eosinophile corpuscles.—These are cells containing coarse, highly refracting granules, which are called eosinophile, or oxyphile, as they stain deeply with eosine and other acid aniline dyes (Fig. 1, *b*). Some of them are of large size, and differ only from the cells of class 1 by the presence of the large eosinophile granules. They are larger than the eosinophile leucocytes of normal blood, and are non-amœboid, or but slightly amœboid. Others of smaller size correspond to the ordinary eosinophile cells of the blood, and possess amœboid movement; various intermediate forms are also present. The total number of eosinophile cells varies much in different cases, but is always increased; whilst the proportion to the total number of leucocytes sometimes exceeds the normal, sometimes not. The relative proportion of eosinophiles in the blood is not to be relied upon as a means of diagnosis in early cases of leucocythæmia, as was at one time supposed, since it is sometimes higher in other conditions. A much more important point is the presence of the above-mentioned large neutrophile and eosinophile cells.

3. The ordinary "multinucleated" leucocytes, or, more correctly, those with multipartite nucleus (as the nucleus is usually single though much lobulated) are also increased in number (Fig. 1, *c*). Along with the cells of class 1 they constitute the great bulk of the leucocytes present. They possess active amœboid movements as in normal blood.

4. Sometimes leucocytes with "mast-cell" granules are present, occasionally in considerable numbers. These granules are a coarse variety, and are stained deeply and of slightly violet tint with methylene blue. They are not found in normal conditions, though they occur occasionally in diseases other than leucocythæmia; their significance is not known. A considerable number of cells with finer basophile granules may sometimes be present.

Such are the main varieties, though some intermediate forms are also found. The small uninucleated leucocytes are not increased in number, and may be diminished; sometimes several fields of the microscope may be examined without any being found.

As abnormal elements in this variety of leucocythæmia we must also mention *nucleated red corpuscles*. They are practically always present, and generally in larger numbers than in any other condition in adult life. Another important point is that their presence does not appear to depend upon the degree of the anæmia, as they may be numerous when the number of red corpuscles is but slightly diminished. They are readily recognised in stained specimens by the very deeply coloured nucleus, and by the perinuclear portion being coloured like the ordinary red corpuscles. The nucleus is usually single and circular, though sometimes it is fragmented, and there may be two or more nuclei of unequal size (Plate, Fig. 1, *d*). The nucleated red corpuscles vary somewhat in size, most being about the size of an ordinary red corpuscle, some considerably larger.

B. *Lymphatic form* (*vide* Plate, Fig. 2).—In this variety the prominent feature is the increase, almost exclusively, of uninucleated leucocytes—lymphocytes and slightly larger corpuscles, such as are normally present in the blood. In the smaller forms the protoplasm is scanty and hyaline; in the larger forms it is relatively more abundant, and often contains small scattered basophile granules. These corpuscles as a rule are non-amœboid, though the larger may show slight movement. In some cases the leucocytes vary greatly in size, from 6 μ to 12 μ, in others they are nearly all under 9 μ. Mitotic figures in these cells have rarely been seen in the blood. The multinucleated leucocytes and the eosinophiles are not at all increased. Nucleated red corpuscles are usually absent, though a few may be found when the anæmia is great. The general appearance of the blood in this form, as seen in a stained film, differs much from that in the previous form (*vide* Plate, Figs. 1 and 2). (A general reference may be made here to art. "Clinical Examination of the Blood," p. 408.)

With regard to the amœboid movements of the leucocytes the general statement may be made that those leucocytes which are amœboid in normal blood are so in leucocythæmic blood also; but in the first form of the disease a large number of non-amœboid cells enter the blood, whilst in the second the cells increased are chiefly of the non-amœboid variety.

In leucocythæmia the *red corpuscles* undergo diminution in number, sometimes to an extreme degree; the number per c.mm. occasionally reaching 1,000,000, or even less. When the anæmia is marked the corpuscles vary in size, both larger and smaller corpuscles being present; they also show irregularity in shape—poikilocytosis. Rouleaux are imperfectly formed in such conditions.

The total number of leucocytes varies much in different cases, and the number relative to the red corpuscles depends also upon the latter. The relative proportion may be as high as 1 : 3, or even higher; and cases have been recorded in which the leucocytes equalled or even exceeded the red corpuscles in number, though most of these observations were made before the hæmocytometer came into use. The total number of leucocytes does not often reach 1,000,000 per c.mm. The number fluctuates considerably from time to time, and occasionally falls greatly. In a case observed by myself (Osler records a similar one) the number fell to normal; but even then the abnormal elements remained in the blood—large uninucleated non-amœboid leucocytes, nucleated red corpuscles, etc.

According to our observations the *blood-plates* are usually very much increased in the spleno-medullary form, sometimes fourfold; in the lymphatic form they are usually diminished in number. We cannot state this as a general rule, as in most cases recorded by others the condition of the blood-plates has not been stated. Nor can we explain this difference in the two types, although the coexistence of increase of blood-plates and increase of multinucleated leucocytes in the spleno-medullary form, as well as in many diseased conditions attended by leucocytosis, is worthy of note.

The *hæmoglobin* is diminished in amount, usually in the same proportion as the number of the red corpuscles, sometimes in rather greater proportion.

Changes in the viscera.—The morbid changes in the viscera are often very extensive, though they vary much in different cases; they chiefly depend upon the following processes:—(*a*) Accumulation and infiltration of leucocytes within organs, leading to enlargement; (*b*) the occurrence of leucocytic thrombosis; (*c*) hæmorrhages, which may be of small or large size; and (*d*) the progressive anæmia which produces fatty degeneration and aids in the production of the general œdema which may be present. We shall afterwards consider whether any of the changes in the organs are to be regarded as primary in nature.

Spleen.—The splenic enlargement is one of the most striking features of the disease; in most cases it is very great, in some cases extreme. The largest spleens are met with in chronic cases; the weight of the organ is often from 5 to 6 lbs., and weights up to 18½ lbs. have been recorded. In the more rapid cases the enlargement is not so marked, and the organ may be less than 1 lb. in weight. The enlargement is generally uniform, so that the form of the organ is maintained; the notches in the anterior border are usually so strongly marked as to be palpable during life. Spleniculi, if present, may share in the enlargement; I have seen in one case a splenículus of 3 inches in diameter. On the surface of the spleen there may be cartilage-like plates of fibrous thickening, or there may be fibrous adhesions. On section the organ may have a fairly uniform red colour varying in depth, and a somewhat dry appearance; or it may contain infarctions of various numbers and ages: sometimes it is studded with them. The infarctions are of different sizes and shapes, being usually more or less wedge-shaped towards the surface, and of irregular outline in the deeper parts; they vary in colour from a deep purple to a pale pinkish gray or yellow, the recent ones being dark in colour. The substance of the organ is usually pretty firm (the more chronic the case the firmer it becomes), owing to a general thickening of the supporting stroma; nevertheless it is often somewhat friable. The Malpighian bodies are, as a rule, indistinct, and it may be impossible to define their outline; sometimes, though rarely, they are very distinct. In the more acute cases, which are often of the lymphatic variety, the organ is generally rather soft, and shows on section a uniform reddish pink colour.

Microscopically, the change is found to consist in a packing of the general pulp with leucocytes similar in character to those found in the blood. Thus in the spleno-medullary form, the large uninucleated cells can be distinguished, and many eosinophile cells also are usually to be found; whilst in the lymphatic form the cells are almost exclusively small uninucleated leucocytes. A general thickening of the reticulum of the pulp may be present in the chronic cases, and thickening of the trabeculæ and vessel walls is also common, the fibrous tissue often showing a hyaline appearance. These latter changes are, however, no doubt secondary to the chronic distension of the organ, aided probably by abnormal metabolic processes; they occur in all conditions of long-standing enlargement of the organ. In the cases which run an acute course, on the other hand, the stroma of the organ may be quite unchanged. The Malpighian corpuscles usually show no alteration; they appear few in number owing to their being separated by the enlargement of the pulp. The infarctions, when present, show the usual minute structure.

Bone-marrow.—As indicated above, the changes in the bone-marrow are of special importance in relation to the pathology of the disease; though further minute histological examination in a large series of cases is still necessary before a very definite opinion can be formed regarding them. According to Neumann, the bone-marrow may present one of two appearances: it may be soft and yellowish white in colour, almost like pus—the "pyoid" condition; or it may be of pinkish colour and firmer consistence—the "lymphoid" or "lymphadenoid" condition. The former is comparatively rare, and has only once been observed by me in a case of spleno-medullary leucocythæmia. In both varieties of the disease the marrow usually presents the appearance described as lymphoid; that is, it has a pale pinkish colour and is moderately firm, though the consistence varies somewhat in different cases. (The term "lymphoid," however, is a bad one, as it naturally suggests lymphoid tissue, from which marrow differs widely both in the cells present and, especially, in the vascular arrangements.) This pale pink marrow fills not only the spaces in the small bones, but also replaces the fatty marrow in the shafts of the long bones, and occasionally causes considerable absorption of the bone. It may be conveniently removed in pieces from the shafts of the long bones, and examined by breaking down a little in normal salt solution tinted with methyl violet, by film preparations, or by means of sections. It is desirable to use all the methods together.

Microscopically, the marrow in the spleno-medullary form is found to contain very much the same cellular elements as are found in normal marrow. The marrow-cells, neutrophile and eosinophile, are very numerous, and, as already stated, closely resemble the cells present in the blood. Nucleated red corpuscles are also fairly numerous, and some of them may be of larger size than usual. Cells containing red corpuscles in various stages of disintegration may also be present, but these are often met with in the marrow in a great variety of other conditions. In sections it is usually found that the fat has been completely replaced by a richly cellular tissue which has the structural arrangements of an active marrow; here, however, there is an excess of the colourless cells. The vascular channels are badly defined, the blood-stream percolating between masses of cells loosely held in position. Giant-cells, generally of smaller size than usual, may be scattered through the section in considerable number. The change may be described in general terms as a hyperplasia of the marrow with excess of the colourless elements. Recently special attention has been directed to the presence of mitotic figures, indicating indirect division of the marrow-cells; and these have been found by some observers to be very numerous. I have found mitoses specially abundant in one case out of four examined—a case of spleno-medullary leucocythæmia in a child aged 14 months. The amount of cellular multiplication taking place, however, at the time of death will probably vary very much in different cases. It would be of great importance to examine a portion of marrow removed during life, but an opportunity of doing so rarely occurs.

In the lymphatic form, in which the marrow may present very much the same naked-eye appearances as in the other variety, there is found a large proportion of small uninucleated leucocytes, which displace to a considerable extent the cells proper to the marrow. The nucleated red corpuscles are few in number. The condition is really a lymphocytic infiltration of the bone-marrow, this tissue being secondarily affected in the same way as other organs (*vide infra*).

Lymphatic glands.—Enlargement of the lymphatic glands is not uncommon if we take all the cases of leucocythæmia. According to Gowers, it occurs in a third of the cases. In most, however, of the earlier cases recorded, the characters of the leucocytes in the blood have not been attended to, and we cannot therefore give statistics of the occurrence of glandular enlargement in each of the two forms of the disease as above defined; though the following general statements may be made. In the spleno-medullary form enlargement of the lymphatic glands is not common. In the majority of cases the disease runs its course without any of the glands being affected; sometimes, however, enlargement occurs, but it usually involves only small groups of glands here and there, and to a small extent. In the lymphatic form, on the other hand, that is when the leucocytes in the blood are of the small uninucleated variety, enlargement of the glands is very common, though by no means invariable. The enlargement may occur early or late in the disease. A single group of glands may show enlargement; usually several groups are affected; more rarely is there a general enlargement. The cervical, axillary, inguinal, and mesenteric glands are most frequently enlarged. The enlarged glands rarely exceed the size of small plums, and usually remain separate and freely movable. They are somewhat soft in consistence, and on section appear succulent and of whitish or slightly pink colour, though there may sometimes be small hæmorrhages into their substance. In the chronic cases some matting of the glands may occur, but this is not the rule.

In the enlarged glands in the spleno-medullary form, collections of cells may often be found towards the periphery, similar to those in the blood and readily distinguishable from the lymphocytes of the adenoid tissue. Their origin is difficult to determine. They may be the result of hæmorrhages, and this would sometimes appear to be the case, red corpuscles being mingled with them; or they may be carried from the tissues by the lymphatics.

In the lymphatic form of the disease the enlargement of the glands is due to an accumulation of lymphocytes, which closely crowd the various parts of the gland and give a uniform appearance throughout. The accumulation is sometimes specially dense in the cortical lymph sinuses. There is usually no trace of thickening of the stroma of the gland, and caseation does not occur unless some other condition be superadded.

Thymus.—Occasionally in lymphatic leucocythæmia the thymus undergoes considerable enlargement and forms a pretty firm mass, somewhat irregular on the surface, in the upper mediastinum. This condition may sometimes be recognised by percussion during life. It may occur in the adult as well as in the young subject. In one case observed by myself, in a woman aged 25, there was great enlargement of the thymus along with enormous enlargement of the spleen, but with scarcely any enlargement of the lymphatic glands. Microscopically, the enlarged thymus shows a well-formed fibrous stroma enclosing pretty large spaces, which are filled with lymphocytes with a small amount of delicate reticulum between them.

Liver.—This organ generally shows some degree of enlargement, and is often 5 or 6 lbs. in weight. In one case at least a weight of over 13 lbs. has been recorded. The enlargement is uniform, the surface usually smooth, and there may be small hæmorrhages under the capsule, though these are not very common. The consistence may be unaltered or may be diminished, and usually the colour is distinctly paler than normal. This pallor may be pretty uniform, but often occurs in pale zones round the portal tracts, thus giving a somewhat nodular marking. Microscopically, in the cases in which the lobules are outlined in this way, there is found an infiltration of the connective tissue of the portal tracts with leucocytes, and the infiltration may extend for some distance into the lobule between the liver-cells and the capillary walls. The infiltration may be pretty general, or it may occur specially in patches here and there. The fibrous stroma, as in the leucocytic infiltrations elsewhere, becomes more delicate, and, as the leucocytes are closely packed together, the appearance is very much as if a growth of lymphoid tissue had taken place round the portal tract. These infiltrations occur especially, though not exclusively, in the lymphatic form of the disease. The capillaries contain large numbers of leucocytes, and some may be plugged by them. Further, in advanced cases there may be a considerable amount of atrophy of the liver-cells. As the result of the anæmia, in many cases these show fatty degeneration, which is usually most distinct in the centre of the lobules. There is no evidence that any cirrhotic change ever occurs as the result of leucocythæmia.

Kidneys.—In the spleno-medullary form the kidneys are usually of normal size and may show nothing abnormal beyond a slight degree of pallor. In other cases the pallor is well marked, and there may be scattered hæmorrhages in their substance or beneath the lining of the pelvis. Occasionally small irregular whitish areas are present, often surrounded by red zones; these are found on microscopic examination to be due to collections of leucocytes in the connective tissue, with a varying amount of hæmorrhage. More rarely there is a diffuse leucocytic infiltration of the connective tissue. The tubules may be normal, but there is very often fatty degeneration of their cells, and occasionally there may be hæmorrhage into their lumen. Sometimes also there are evidences of catarrh. The glomeruli are usually normal, but hæmorrhage within the capsule of Bowman is sometimes met with.

In the lymphatic form of leucocythæmia the connective tissue of the kidneys is not infrequently the seat of a diffuse lymphocytic infiltration which may lead to great enlargement. In the case of a boy aged eight, reported by Dr. John Thomson and myself, each kidney weighed 16¼ ounces, and the left kidney was easily palpable below the spleen during life. The enlargement usually affects both cortex and medulla in a uniform manner and in equal proportion. The tissue is pale and the markings are regular, though there may be small hæmorrhages here and there. The consistence may be nearly normal, or it may be distinctly soft, so that the kidney substance bulges somewhat when the section is made. Microscopically there is found in these cases simply an enormous infiltration of lymphocytes in the connective tissue of the organs, so that the tubules and other elements become widely separated from one another. The tubules themselves may remain normal, or any of the conditions mentioned above may be present.

Occasionally infarctions are found in the kidneys as the result of leucocytic thrombosis, but these are rare.

Other organs, such as the *suprarenals*, *thyroid*, *ovaries*, etc., may show enlargement of the same nature as that of the kidneys, though they are less frequently affected. When such affection is present their tissue becomes softer; and has usually a diffuse pinkish colour, the normal markings being somewhat blurred.

In most of the cases in which such diffuse infiltration of the connective tissue of organs occurs, the disease runs a more or less acute course.

Bizzozero has observed numerous mitotic figures in the leucocytes infiltrating the tissues, and Hindenburg found them in the leucocytes in the spleen pulp, in the capillaries of the liver, and in the sinuses of lymphatic glands, but not specially in the germ-centres of lymphoid tissue.

Alimentary canal.—The lymphoid follicles in connection with the various parts of the alimentary canal may undergo enlargement in the lymphatic form of the disease, and there may be in addition more diffuse leucocytic infiltration of certain parts. This latter may occur in the tissues of the gums, leading to swelling which may be followed by ulceration. The tonsils in some cases may undergo considerable enlargement, and the lymphoid tissue of the pharynx and neighbouring parts may be similarly affected. The solitary glands in the stomach have also been found enlarged in a few cases. In the intestines the changes are occasionally of a striking character. Swellings of considerable size may be produced by enlargement of the Peyer's patches or solitary glands, or by irregular leucocytic infiltration of the mucous membrane. Such changes may be found both in the large and small intestine, but usually one part of the intestine is affected in a special degree. The swelling may be followed by ulceration, which is usually irregular, though the ulcers in some cases have been described as "typhoid-like." Along with these intestinal changes there is usually enlargement of the mesenteric glands, though this latter may occur independently of any affection of the intestines. An "intestinal" form of leucocythæmia was described by Behier from the condition just described, but it is simply a variety of the lymphatic form; different organs being affected very variously in cases of the disease.

Heart.—Fatty degeneration of the muscular fibres of the heart is often present, and, in cases where there has been marked anæmia, the inner surface of the organ may show extensive pale yellowish mottling. The organ often contains yellowish white coagula which, owing to the large number of leucocytes contained in them, may appear as if pus were mixed with the fibrin—a condition which attracted the attention of earlier observers. As a rare condition may be mentioned the occasional occurrence of patches of *myomalacia cordis* in the heart wall, the results of thrombosis of the branches of the coronary arteries. We can find no evidence that any hypertrophy of the heart takes place as the result of leucocythæmia, though some writers mention its occurrence. If present, it is due to some coexisting complication.

Lungs.—In the lymphatic form of the disease the connective tissue of the lungs may be the seat of leucocytic infiltration. The walls of the bronchi and the peribronchial tissue are chiefly affected, and the condition may be diffuse or localised so as to form thickenings. This change, which may be found only on microscopical examination, is of the same nature as that occurring in the connective tissue of other organs. On microscopic examination also many of the small vessels may be found plugged with leucocytic thrombi, and hæmorrhages may be seen around them. The lungs are generally œdematous, and various other conditions may be present as complications.

Hæmorrhages.—Small hæmorrhages have already been mentioned as occurring on the surface or in the substance of various organs; and they are also common on serous membranes generally, in the periosteum, and in the skin. Hæmorrhages of larger size may be found in various parts; sometimes they are apparently spontaneous, sometimes produced by slight traumatism; and often they take place from mucous surfaces. Special mention must be made of the occurrence of cerebral hæmorrhages on account of their importance, as they are not infrequently the direct cause of death. The hæmorrhage is sometimes single and of large size, tearing up the brain substance to a great extent, and may occur in any part. It is usually progressive and leads to a fatal result, though occasionally arrest and recovery may take place. Sometimes multiple hæmorrhages are found, as in one case observed by myself, in which there were fully a dozen hæmorrhages of various sizes in the cerebrum and cerebellum, both in the superficial and in the deep parts. These hæmorrhages are almost certainly the result of leucocytic thrombosis occurring in badly nourished vessels, the thrombosis probably beginning in the small veins. Many of the small vessels in the neighbourhood of the hæmorrhage may be found plugged in this way, and small hæmorrhages with large numbers of leucocytes may be seen in their perivascular sheaths.

Organs of special sense.—The eye and ear may be affected in like manner; namely, by the occurrence of hæmorrhages, and of leucocytic infiltrations of their tissues. In the retina minute hæmorrhages are of common occurrence, and are often associated with leucocytic infiltrations along the lines of the vessels and in patches—the condition described by Liebreich as *retinitis leucæmica*, though it is not really of inflammatory nature. Occasionally a more diffuse leucocytic infiltration of the layers of the retina has been found. Hæmorrhage into the vitreous is of rare occurrence. Similar infiltrations have been found in the structures of the inner ear, and have been associated with subjective symptoms, such as vertigo; in one or two cases hæmorrhage has been found as the cause of sudden deafness.

PATHOLOGICAL CHEMISTRY.—At a comparatively early date chemical analyses were made of the blood and organs in leucocythæmia, but many of the results are vitiated to a considerable extent by the fact that the material used was obtained after death, and therefore at a time when important changes had been brought about by bacterial action. In some cases, however, analyses have been made of the blood obtained by venesection, and of the spleen excised during life. The statement formerly made, that the blood has an acid reaction, depended upon examination of blood in which acidity had been produced by post-mortem change, and is incorrect; though the alkalinity is usually diminished. The fibrin has been found increased in amount, though coagulation takes place slowly—a circumstance which has been ascribed by some to the presence of peptone in the blood, though this has not been certainly proved. Matthes found deutero-albumose in the blood taken fresh, but no peptone.

As the result of many independent analyses, xanthin bases have been found in increased amount. Salomon found that hypoxanthin forms in ordinary blood after it has been allowed to stand; but in fresh leucocythæmic blood, obtained by venesection, a considerable quantity is present. Further, more hypoxanthin has been obtained from post-mortem specimens of blood in leucocythæmia than under other conditions. According to most authors, uric acid is not found in the blood, though some have asserted its presence. The presence of certain organic acids—lactic, formic, and succinic—has also been affirmed, the first-mentioned being found by Salomon in the proportion of ·05 per cent in fresh blood. Lactic acid, again, is formed in normal blood when taken from the body, being due, according to Salomon, to a fermentative change in the leucocytes; and one would expect this post-mortem formation to be increased in leucocythæmia. By some observers, other substances—gluten, leucin, nucleo-phosphoric acid, guanin—have been found as abnormal bodies or increased in amount, but chiefly in specimens obtained after death.

Though there is probably no chemical substance in the blood peculiar to the disease, the increase in the xanthin bases, discovered many years before any definite opinions were formed regarding their origin, is a well-established fact. According to the view which has recently obtained pretty general acceptance, these bodies are formed from leucocytes, and rather from their breaking down than as a product of their metabolism. According to Kossel, their chief source is the nuclei of these cells. Horbaczewski has also traced the formation both of the xanthin bodies and of uric acid to the same source. He found that from portions of spleen outside the body, by varying the conditions, he could at one time obtain uric acid, and at another xanthin compounds.

One more point of considerable interest is the occurrence in the blood and organs after death of the minute crystals known as Charcot's crystals. They are not present in the fresh blood, but may be found after it has been kept for some time. They are specially abundant in the spleen and in the bone-marrow; and, according to Neumann, they are present specially in the spleno-medullary form, being usually absent in the lymphatic form. They are usually regarded as a post-mortem product, though not necessarily produced by decomposition; yet Westphal found them in blood taken from the spleen during life and examined at once on a warm stage: hence he concludes that, in the spleen at least, they may be present during life. They are not peculiar to leucocythæmia, but may be found in the marrow in other conditions; and, as Leyden first discovered in the case of bronchial asthma, they may be found in the sputum. They are minute, elongated, symmetrical octahedra, and usually measure 10 μ in length; though smaller and larger forms are also found. They are soluble in warm water and in solutions of alkaline carbonates, very sparingly soluble in cold water, and insoluble in alcohol, ether, and chloroform. There is some doubt regarding their exact constitution, but at any rate they contain phosphorus; according to Schreiner, they are a compound of phosphoric acid and a base "spermin" which has the formula

C_2H_5N. They are probably the result of cellular disintegration also, the conditions under which they are found tending to support this view.

Chemical examination of the organs has given results somewhat analogous to those described above. Both in the liver and in the spleen, obtained after death, various observers have found a considerable quantity of peptone, also of xanthin bodies (especially of xanthin itself, hypoxanthin being less abundant or absent), also of organic acids, especially lactic, formic, and succinic, and leucin and tyrosin in small and varying amounts. In most analyses uric acid has not been found. Bockendahl and Landwehr obtained from a leucocythæmic spleen excised during life—peptone, 1 per cent; lactic acid, ·012 per cent; succinic acid, ·002 per cent; xanthin, ·038 per cent: leucin was present, but no tyrosin, uric acid, or glycogen.

The amount of iron in the liver and spleen has been estimated in a few cases, and has been found somewhat increased; v. Bemmelen found a proportion of 0·22 to 0·27 per cent of dried substance in the liver, and Prof. Stockman 0·337 per cent in the liver and 0·29 per cent in the spleen. Stockman attributes the increase in his case to the numerous hæmorrhages in the body. Granboom also found more iron in the liver in leucocythæmia than in a number of other diseases investigated; namely, 0·09 per cent of liver substance (not dried). The liver-cells, however, do not usually contain pigment granules which give the iron reaction, the presence of which is such a striking feature in pernicious anæmia.

The general result of chemical investigation in the disease has then not been to reveal any very striking change in metabolism; the various chemical substances found in the blood, organs, and also in the urine (*vide infra*) being, so far as evidence goes at present, chiefly the result of excessive disintegration of leucocytes. As the number of leucocytes in the body is greatly in excess of the normal, and as these cells have probably a comparatively short life, the amount of leucocytic destruction must be greatly increased and accordingly the products of their disintegration also.

CONDITIONS OF OCCURRENCE AND REMOTER CAUSES. — Leucocythæmia may occur practically at any time of life; but is most common in middle adult life—from thirty to fifty years of age. The results of statistics independently compiled agree in showing that it is twice as common in man as in woman. Cases are most numerous about the age of thirty in man and forty in woman, but it appears to affect men at a greater age than women, being exceedingly rare in the latter after sixty, while a considerable number of cases have been recorded in men above seventy. The disease is, however, more common in children than was formerly supposed; probably many cases have been overlooked. A greater proportion of cases of the lymphatic type occurs in the early years of life than of the spleno-medullary; but both varieties may affect children a few months or even weeks old. It is found in people of all classes of society, its occurrence being apparently little affected by the conditions of life and surroundings; though it is sometimes stated to be more common in the poorer classes. It appears to occur in various countries with much the same degree of frequency: statistics of the proportion of cases of leucocythæmia to the total number of cases in various Continental hospitals for ten years, given by v. Limbeck, show a considerable difference in different towns; this difference, however, may be accidental, as it is met with in the case of towns not far distant from one another. From this table the average proportion of cases of leucocythæmia to other cases is about 3 to 10,000.

Hereditary influences appear to play little or no part in the proclivity to the disease. Only a few cases are on record in which more than one member of the same family have been affected by the disease. Such cases have been recorded by Chambers, Senator, and Eichorst. Senator, quoted by Eichorst, observed the disease in twins. Instances in which one member of a family has suffered from leucocythæmia and another from splenic or glandular enlargement are also few in number, and occurrence of leucocythæmia in one of the parents and in one of the children of the same family is almost unknown. Leucocythæmic women bear children free from the disease; on the other hand, the child of a healthy mother may show the disease when but a few weeks old.

As *remoter causes* syphilis, rickets, rheumatism, acute febrile diseases, depressing mental conditions have been mentioned by writers on the subject, but these would probably be found to be the most common antecedents of a large number of cases of any chronic disease. With regard to malaria, however, there does appear to be some connection more than accidental. Sir W. R. Gowers found a history of previous intermittent fever in a fifth of a number of cases, and exposure to malarial influence in a quarter. Though his results have not been entirely confirmed by the statistics of others, still a malarial history appears to be too common to be regarded as a mere coincidence. We cannot, however, go beyond this, that malarial fever probably acts as a disposing condition. It is also to be remarked that the protozoon, now sufficiently established as the cause of malaria, has not been found in the blood in cases of leucocythæmia. In many cases of acute leucocythæmia there has been a history of pre-existing inflammatory or ulcerative conditions about the mouth, fauces, or intestine; but it is possible that these were early signs of the disease itself. A history of a blow over the spleen, or of injury to the bone, has been noted in one or two cases, but the coincidence must be regarded as accidental.

In woman sexual processes have been regarded by many as having an etiological relation to the disease, which has frequently been observed to start during pregnancy, after parturition, and, especially, during a prolonged lactation. Without denying that these processes may have some relation to the disease, still, in view of the considerable proportion of the adult life of a woman which on an average is thus occupied, and of the fact that at such times any abnormal condition is more likely to be noticed, I think that the connection is only accidental.

We may summarise our knowledge regarding the conditions of occurrence by saying that leucocythæmia may occur at any age; that no connection with the surroundings and conditions of life of the patient can be traced, and that, with the possible exception of malaria as a remote cause, no relation to any previous disease has been established. In the great majority of cases the individuals affected had previously been in good health.

Further—and this is a fact of importance—the disease is one which affects the lower animals; cases have been observed in the dog, cat, ox, sheep, pig, and others.

NATURE AND ETIOLOGY.—It is evident that in the case of a disease such as leucocythæmia, in which there is so marked an alteration in the corpuscular elements of the blood, any conclusion regarding its nature must accord with the known facts of the formation and destruction of the corpuscles. As there are few subjects on which there has been so great a diversity of opinion, it would be quite out of place here to discuss the various hypotheses in detail. I shall, therefore, only state the inferences which appear to be justified from a consideration, on the one hand, of the changes in the blood and organs; and, on the other hand, of the views now most widely accepted regarding the life-history of the blood corpuscles.

If we consider first the lymphatic form of leucocythæmia, as being probably the simpler, we find that the essential change is the presence of enormous numbers of small uninucleated leucocytes throughout the body; both in the blood and in various tissues. Such a condition, in view of its nature and extent, can only, I think, be due to an excessive and apparently purposeless proliferation of these cells. And, further, all the histological changes present can be explained by such a proliferation. In fact, the condition is closely allied in nature to tumour growth, to sarcoma, for example; and the diffuseness of the lesions would be explained by the characters of the cells involved, these being normally present throughout the tissues, and constantly in movement. Hence instead of distinct, tumour-like masses, we find diffuse infiltrations of the tissues, leading to uniform enlargement of organs. The anæmia may be explained by the infiltration of the hæmopoietic tissue of the bone-marrow, and by the gradual diminution of the blood-forming area. As already stated, we cannot infer the origin of the disease from the characters of the cells; we can only judge roughly from the organ or tissue which first shows enlargement. One of the striking features of the condition is that in different cases the various organs are affected in a great variety of ways, and this cannot as yet be explained; though there are analogous facts in the case both of the infective granulomata and also of malignant tnmours.

In the other, the spleno-medullary form, the character of the cells in excess suggests an origin in the bone-marrow. The large uninucleated cells in the blood correspond with the marrow-cells or "myelocytes," the large eosinophile cells in the blood with the eosinophile marrow-cells; whilst the nucleated red corpuscles, which are usually numerous in the blood in this condition, are in the normal state only found in the bone-marrow of the adult. No doubt, in other abnormal conditions the nucleated red corpuscles pass into the blood, but never in such numbers as in this form of leucocythæmia, nor when the degree of anæmia is so slight as it may be in this disease. It would appear, in fact, as if there were an extension of the cells of the marrow into the blood.

The evidence of excessive division of these cells as shown by mitotic figures in the bone-marrow and also in the blood, is not quite conclusive; but it must be remembered that the disease is usually a chronic one, lasting sometimes for several years, and that there also appear to be remissions, if we may judge by the number of leucocytes in the blood. We may therefore regard the hypothesis of excessive proliferation of the cells in the marrow, with an extension of these cells into the blood, as being that which is most in accordance with facts.

I cannot see sufficient evidence in support of the view held by many authors that leucocythæmia is primarily a disease of the spleen. This organ shows merely a distension of the pulp with leucocytes, and the result of that distension when chronic, namely, thickening of the stroma; and the reason that it is almost invariably enlarged is probably to be found in the relation of the circulating blood to the spleen pulp. Besides, the spleen undergoes enlargement (though usually to a less degree, owing probably to the shorter duration of the disease) in the lymphatic form as well as in the spleno-medullary. Unless, therefore, we are to assume that in all cases where the spleen undergoes great enlargement it is the primary seat of the disease, it must be admitted that great enlargement may occur secondarily. There is, besides, evidence that in normal conditions leucocytes break down in the spleen, and the enlargement may really be the result of an attempt to deal with the abnormal supply of leucocytes. In malaria we have a striking example of the degree which splenic enlargement may reach as a secondary affection. We cannot infer a primary affection of the organ from great enlargement.

The view that the disease essentially consists in an excessive proliferation, in the one variety, of the small uninucleated leucocytes, in which case the proliferation may start in various organs, and, in the other variety, of the cells of the marrow, is the one most in accordance with the microscopical changes in the blood and various organs. It is also in accordance with the views generally accepted by recent authorities on the relations of the leucocytes to the red corpuscles. According to these views, with which, from my own observations, I fully agree, none of the leucocytes of the blood becomes transformed into red corpuscles, these being formed from special cells—"erythroblasts"—in the bone-marrow. The leucocyte is a distinct kind of cell which has a life-history of its own, and special functions. The so-called "marrow-cells" are merely a variety of large leucocytes which by their division produce smaller leucocytes, which afterwards appear in the blood. I accordingly consider that the view, still held by many pathologists, that in leucocythæmia there is an interference with the transformation of leucocytes into red corpuscles, has no real basis. It is also to be noted that in normal conditions the youngest form of leucocyte and that which shows most active proliferation is the lymphocyte, whilst the marrow-cells form an older series of cells in which division is less active. In the form of leucocythæmia in which those lymphocytes are in excess, the disease usually runs a more rapid course.

With regard to the chemical changes in the blood, various organs, and urine (*vide infra*), I have already stated that probably all can be explained by the excessive disintegration of leucocytes which must occur in the disease.

When we come to inquire into the immediate cause of this proliferation of leucocytes, we find that there is as yet little ground to go upon. Naturally two hypotheses present themselves; namely, that it is due to some microparasite, or that it is of the same nature as tumour growth, whatever that may prove to be. There are facts to support each, but neither is more than a hypothesis. No parasite of the nature of the malarial organism has been observed in leucocythæmia, and there is no adequate evidence that any bacterium is concerned in the disease. Attempts have been made to transmit the disease to lower animals by injecting either the fresh blood or the juice of a recently excised leucocythæmic spleen; but they have been without positive result.

It has recently been suggested that the excess of leucocytes may be due to the continued presence in the blood of some chemical substance, such as in normal conditions produces an increase of leucocytes. Vehsemeyer, for example, has attempted to produce the disease by often-repeated injection of peptone, and has found a very considerable increase of the leucocytes, lasting for several weeks. This, however, is only a continued *leucocytosis*, not leucocythæmia; the leucocytes differ in the two conditions (*vide* p. 661), and in the former there is no affection of organs such as occurs in the latter. Leucocythæmia has never been produced experimentally.

In the absence of knowledge regarding the agent producing the excessive proliferation of leucocytes, we cannot definitely assign the place of leucocythæmia in the category of disease. On the whole it presents most points of analogy to the growth of tumours, the analogy being specially striking in the lymphatic variety; but, on the other hand, it is not absurd to suppose that it may yet prove to be due to a microparasite.

SYMPTOMS.—In describing the symptoms of leucocythæmia, we may distinguish an acute and a chronic form;[1] these are fairly well defined, though cases of intermediate character occur. I shall give an outline of the course of the disease in the two forms, taking first the chronic form, which is the commoner.

The onset of the disease is generally gradual and insidious. In many cases the earliest symptoms are produced by the splenic tumour; a dragging sensation or pain in the left hypochondriac region or a general swelling of the abdomen may first be complained of. In others, weakness, breathlessness on exertion, giddiness, or gastric symptoms are the first indications. Sometimes hæmorrhage from the nose, more rarely from the bowels, first leads the patient to seek advice. At this stage the patient usually looks in pretty good health, and has not lost flesh, though a certain degree of pallor may be present. Examination of the blood may show a moderate or great increase of the leucocytes (for characters, *vide* p. 637), and the red corpuscles may be only slightly diminished. The spleen, even at this early period, may show enormous enlargement, and its lower margin may be at the iliac crest. The changes in the blood and the condition of the spleen usually render the diagnosis easy. If the temperature be taken regularly, slight irregular pyrexia may often be detected. This generally occurs at night, the temperature rising a degree or more on some days, with intervals of a normal condition; it may be accompanied by sweatings; though, independent of rise of temperature, such a tendency to sweating is not an uncommon symptom. Disturbances of the alimentary system often appear, vomiting or diarrhœa from time to time being not infrequent.

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[1] These two forms only approximately correspond with the lymphatic and spleno-medullary forms (*vide infra*).

Such are the common symptoms in the early stages of the disease; and in distinctly chronic cases patients may remain in pretty much the same condition for months, or even for one or two years. In some cases, in fact, they may enjoy tolerably good health with the leucocythæmic condition of the blood well marked and the spleen of great size. More frequently the general health is considerably impaired, more prominent symptoms occurring at intervals and tending to become aggravated. In this stage, under suitable treatment, considerable improvement in the general health may take place, and the number of leucocytes may even diminish considerably. Periods of relapse, however, follow, and in the course of time a greater or less degree of cachexia usually supervenes. Pallor and breathlessness become more marked, the pulse is often feeble and rapid, the temperature is more frequently elevated and still shows the same irregular character. The abdomen may show considerable tumidity, owing partly to the splenic enlargement, partly to chronic flatulent distension, and partly to ascites, which is no uncommon condition. The patient loses flesh, becomes more and more asthenic, and is confined to bed. Even in this stage a certain amount of improvement may occur, but too often the course is steadily downhill. A tendency to hæmorrhage, if not present before, often appears now, and in this way the prostration is increased.

A fatal termination may be brought about in various ways. In many cases advancing cachexia and anæmia are followed by the occurrence of general dropsy, which gradually increases, and the patient dies from heart failure with pulmonary œdema. This, indeed, is the usual sequence of events unless some fatal complication occur. In other cases severe hæmorrhage from the nose, bowels, or elsewhere may be the immediate cause of death; and in a certain proportion of cases death is produced suddenly by the occurrence of single or multiple hæmorrhages in the brain. Occasionally severe diarrhœa contributes largely to the fatal termination; in other cases intercurrent affections, such as pneumonia or peritonitis.

Such, in outline, is the course of the disease in its chronic form, and most cases of spleno-medullary leucocythæmia in adults conform to this description. The disease in this form usually lasts for from one to two years after the first symptoms, though a longer duration is not uncommon. After distinct cachexia sets in, the fatal result generally follows in a few months, though it may occur at any time.

In another group of cases the disease runs a much more rapid course, and to these the name **acute leucocythæmia** has been given, though it has only a relative significance. Leucocythæmia is more apt to have this character in the earlier years of life, especially when the disease is of the lymphatic variety. Of 17 acute cases collected by Ebstein, in only 7 were the patients over thirty years of age; and in 4 cases observed by myself the greatest age was twenty-six. In some such cases a fatal result may follow as early as four or five weeks after the first noticeable symptoms, or even earlier; how long after the beginning of the disease we cannot, of course, say. The characters of the disease are of the same nature as in the chronic form, but are exaggerated in degree and in rapidity of course. Rapidly advancing pallor and weakness, or severe hæmorrhage, may be the first indications of the disease. Irregular pyrexia, often with great perspiration, thirst and anorexia, vomiting, diarrhœa, repeated bleedings from the nose, gums, or bowels, and subcutaneous extravasations, are amongst the most usual symptoms during its course. Enlargement of the lymphatic glands is sometimes well marked, and may be one of the earliest changes to be noted by the patient. Death may be preceded by a typhoid-like condition; sometimes it results from general œdema and heart failure, sometimes directly from hæmorrhage. In such acute cases the splenic enlargement is usually only moderate in degree, or may even be slight; though the increase of leucocytes is generally great and the anæmia sometimes extreme.

After this outline of the main features of the disease the more important clinical conditions may be described in greater detail.

The condition of the *blood* is always of importance, and when examined from time to time gives valuable indications as to the course of the disease. In the chronic cases the number of leucocytes often remains about the same for a considerable period of time, though slight fluctuations occur. Their number, however, varies much in different cases. In one case, for example, the leucocytes may number 200,000 per c.mm., in another 600,000 per c.mm.; and when an examination is made some months later the numbers in the two cases may be little altered. It is not the rule to find a gradual increase in the number in proportion to the duration of the disease, although when the condition of the patient grows worse the number of the leucocytes often increases. Occasionally, under treatment, the leucocytes may become considerably diminished, and may even fall to normal. It is not possible, however, to say that the patient is cured, though the diminution is usually accompanied by an improvement in the general health; the abnormal elements remain in the blood, and the splenic enlargement is sometimes little altered; though sometimes it is considerably diminished. In the more acute cases a considerable augmentation in the number of leucocytes may be observed in the course of the disease, and, as there is usually much diminution of the red corpuscles at the same time, the proportionate increase is more marked still. In a rapid case observed by myself, the numbers changed from leucocytes 209,000, red corpuscles 2,085,000, to leucocytes 379,000, red corpuscles 902,500, in less than four weeks. Throughout the greater period of the disease in the chronic type, the red corpuscles usually number about 3,000,000 per c.mm., their number remaining almost stationary for a considerable time, though falling considerably towards the close of the disease. It is always a grave sign when the number of the red corpuscles steadily diminishes in spite of treatment. Moreover, it should be borne in mind that when the condition of the blood is stationary, or even improving, a rapid aggravation leading to a fatal result may set in at any time.

The *splenic enlargement* corresponds in general characters with that met with in other conditions. It is greatest in long-standing cases, and may exceed that met with in any other disease. The enlargement, for anatomical reasons, extends mostly forwards and downwards; but sometimes, when the downward extension is interfered with by adhesions or by a powerful costo-colic ligament, the extension upwards is very marked. The lower margin may be as low as the anterior superior iliac spine, or even lower; whilst the anterior border may reach beyond the middle line, occasionally even as far as the anterior superior iliac spine on the right side. The form of the organ is maintained, and, as its consistence is usually firm, its rounded margin can be readily palpated, the notches in the anterior margin being often well marked. The enlarged spleen often gives rise to a sense of dragging or heaviness, the uneasiness being increased after food; and sometimes, owing to the occurrence of perisplenitis, actual pain of a dull or sharp character may be present, especially on movement. It may also interfere to a varying extent with the movements of the diaphragm, and complicate respiratory troubles. When such great enlargement has been reached, the size remains as a rule fairly constant, showing only slight variations from time to time. Sometimes, however, considerable diminution takes place, which may or may not be accompanied by an improved condition of the blood. In the more rapid cases of leucocythæmia the spleen may extend but little beyond the costal margin; and, as its consistence is less firm, palpation of its border is not so readily effected. Between the size of the spleen and the number of leucocytes in the blood there is no fixed relation. I have seen in the more acute cases an enormous excess of leucocytes with but moderate splenic enlargement; and, on the other hand, I have seen the number of leucocytes in chronic cases fall to a little above normal while the spleen remained of very great size.

The *lymphatic glands*, when enlarged, may give rise to considerable swellings which are readily visible; in other cases the condition is discovered by palpation. The anatomical changes have already been described (*vide* p. 642), and the clinical characters correspond. The enlarged glands are usually free from matting or induration around, are neither painful nor tender, and may show considerable fluctuations in size from time to time. Occasionally an area of dulness can be determined during life over the upper part of the sternum, which is due, not to enlarged lymphatic glands, but to a diffuse lymphoid infiltration of the thymus or its remains. Pressure symptoms are rarely produced. I repeat that the lymphocytes may be in great excess in the blood whilst glandular enlargement is slight or even absent. In one case of this nature recently observed by myself there was extensive leucocytic infiltration of the liver, kidneys, and suprarenals, with considerable enlargement of the spleen; the lymphatic glands being almost unaffected.

The changes in the *bone-marrow* are usually unaccompanied by any symptoms. Mosler was the first to describe tenderness over the sternum as a symptom in the disease: this he found to be due to an overgrowth of the marrow, with absorption of the bone; and a like condition has been noted in other cases. Occasionally there is a dull pain in addition to tenderness, and these symptoms may be present in other bones besides the sternum. Such symptoms are, however, the exception rather than the rule, and it may be definitely stated that an extensive hyperplasia of the marrow may be present without any subjective indication whatever. In a few acute cases of the lymphatic type similar tenderness over the bones has been noted, so that its presence does not necessarily indicate a primary change in the marrow.

The *thyroid*, when it is the seat of leucocytic infiltration, may be obviously enlarged during life. I have only once observed this, a symmetrical enlargement of moderate degree and painless, occurring in a case of acute lymphatic leucocythæmia. So far as I can ascertain, no symptoms referable to the suprarenals occur when these are the seat of leucocytic infiltration.

Disturbances of the *alimentary system* are common, especially in the more acute cases, and may give rise to most troublesome symptoms. The tonsils and lymphoid tissue of the pharynx may be enlarged and interfere somewhat with deglutition, especially when an inflammatory condition is superadded, as is sometimes the case. The enlargement, as in the case of the lymphatic glands, may show fluctuations from time to time. In such cases the condition first described by Mosler as leucæmic stomatitis is apt to occur also—a condition in which the gums and other parts of the mouth become swollen, inflamed, spongy, and sometimes ulcerated; it is often attended with bleeding. The change somewhat resembles that found in scurvy, and there is often decomposition of the secretions and blood, with marked fœtor. In a few cases gangrenous processes have supervened.

The appetite varies considerably. In the earlier stages in chronic cases it is usually little if at all impaired; in a few cases it has been described as unusually great. Discomfort after a full meal is a common symptom, and is to be ascribed in part to the pressure of the enlarged spleen on the stomach. In the later stages of the disease, when there is cachexia, and especially in the acute cases, gastric symptoms may be very prominent. There is complete loss of appetite, very feeble digestive power, vomiting, and occasionally hæmatemesis; though bleeding from the stomach is not so common as from the nose or bowels, and usually occurs only late in the disease.

Intestinal symptoms are comparatively common. There may be flatulent distension and constipation alternating with diarrhœa; a tendency to the latter is often well marked throughout chronic cases. But diarrhœa is sometimes severe in degree, especially in the stage of cachexia; and it may largely contribute to a fatal result. It is sometimes accompanied by tenesmus and by bleeding from the bowels, the bleeding varying greatly in amount, but being sometimes profuse and occasionally the cause of death. In such cases often no lesion of the intestinal mucous membrane can be found after death, there being apparently a general oozing of blood from its surface; occasionally with the lymphatic variety of the disease the lesions above described are found associated.

Acute peritonitis may supervene and determine a fatal issue. The cause of the condition is doubtful, but in the cachexia towards the end of life micrococci may gain entrance to the blood and lodge in the spleen, as was found by myself in one case; and it is possible that thence they may pass to the surface of the organ and infect the peritoneum. In other cases peritonitis may be set up by the process of tapping.

Enlargement of the liver can often be ascertained by percussion, and its lower margin is sometimes palpable; but usually no symptoms are produced by the affection of this organ. Jaundice is not met with, unless as the result of some superadded condition. Great leucocytic infiltration of the portal tracts may, however, possibly aid in the production of ascites, which is often present towards the close of the disease. The ascites may occur as part of a general dropsy, but sometimes the effusion into the peritoneum is well marked when there is little or no dropsy elsewhere, and may require repeated paracentesis. Spontaneous hæmorrhage into the peritoneum has been described, but is a very rare occurrence.

The symptoms in connection with the *circulatory* and *respiratory* systems are mostly referable to the general condition, and especially to the anæmia. Palpitation, breathlessness on exertion, giddiness, and the like tend to become worse as the disease advances. The pulse becomes softer and more rapid, but is usually regular, even in the later stages of the disease, when, owing to the fatty change which is often present, the heart's action may be very feeble. Systolic hæmic murmurs may be heard over the heart, and a bruit over the veins at the root of the neck. The heart is sometimes displaced upward and slightly to the right side by the splenic enlargement and the abdominal distension. Dyspnœa is often a distressing feature in the late stages of the disease, even to the full extent of orthopnœa. Several factors are concerned in the production of this symptom. In addition to the anæmia present and the feeble action of the heart, effusion into the pleural cavities may largely contribute to it, and the condition is aggravated by the abdominal distension which displaces the diaphragm upwards and restricts its movements.. Œdema of the lungs usually precedes death, which may come about very gradually. In one case, observed by myself, in which death took place somewhat suddenly, there was extensive leucocytic thrombosis in the small pulmonary vessels, along with large pale coagula in the large trunks. Bronchial catarrh is not uncommon throughout the disease, and the cough, in some cases very troublesome, is attributed to reflex causation. Pleurisy and pneumonia may be mentioned as complications.

Dropsy is common in cases in which there is advancing cachexia; it results from the anæmic condition, general malnutrition, and gradual heart failure. Anasarca may be of extreme degree, the epidermis may be raised in blebs, and an erysipelatous condition sometimes supervenes. Effusions into the various serous cavities are common, and, as I have said, ascites is often considerable.

Hæmorrhage into the tissues, or from mucous surfaces, occurs, at some period of the disease, in the majority of cases. Of all the varieties of hæmorrhage epistaxis is the commonest. It may occur at any period, and is not uncommonly an early symptom. It may recur frequently throughout the disease and be moderate in degree; sometimes it is very severe and may be the cause of death. Hæmorrhages from the stomach or from the bowels, though less frequent than epistaxis, are by no means uncommon, those from the bowels being the commoner. The amount and frequency of the hæmorrhages vary much in different cases. In the case of intestinal bleeding, for example, there may be only a small amount of altered blood in the stools; the fæces may be pulpy and contain a considerable admixture of blood, or almost pure blood may be passed from the bowel. Hæmorrhages from the lungs and kidneys and from the female genital tract are rarer events. Petechiæ in the skin may occur, but usually only in the advanced stages of the disease; sometimes in the more rapid cases, often of the lymphatic variety, the skin hæmorrhages may be much larger and of more diffuse character, as in purpura hæmorrhagica. Hæmorrhage into the joints has also been recorded. Hæmorrhage into the deeper tissues, or muscles, is another complication, sometimes resulting from slight traumatism, sometimes apparently spontaneous. I have seen more than a pint of blood effused into the abdominal muscles, as the result of paracentesis when the puncture was made a little to one side of the middle line. Hæmorrhage into the brain has been mentioned above as a not infrequent cause of death. Fatal cerebral hæmorrhage may occur suddenly, or may be preceded by symptoms, as in the case of smaller initial hæmorrhages. As the hæmorrhage is in some cases multiple and in other cases very extensive, localisation during life is usually very difficult. In giving a prognosis in cases of leucocythæmia, the possibility of the occurrence of cerebral hæmorrhage should be kept in view.

Elevation of the *temperature* at some period of the disease is almost invariable. In the early stages in chronic cases slight irregular elevations, more marked at night, may occur from time to time, with periods of normal temperature between. In the later stage, and especially in cases running an acute course, the pyrexia is more marked, though still showing an irregular character. The temperature sometimes reaches 102° or 103° at night, and falls a degree or two in the morning; though sometimes it shows a more continuous rise. Occasionally slight rigors occur with the rise of temperature, the causation of which is obscure.

The *urine* is generally normal in quantity, though towards the end of the disease it may be diminished. Its specific gravity varies, but is usually pretty high; an acid reaction is usually well marked. The amount of urea has been found to vary in different cases, though it is often little altered; but increase in the quantity of uric acid, observed by Virchow at an early date, is an almost invariable occurrence. The amount of the latter has been recorded as reaching over 3 grms. a day, but more recent analyses show that it rarely exceeds 1·5 grm. A deposit of urates often appears in the urine after standing, and uric acid crystals may also be found. The xanthin bases, of which traces are found in normal urine, are also increased in amount, and some of the rarer members of the series—heteroxanthin, guanin, etc.—have been found by different observers. Bondsynski and Gottlieb, in a case of spleno-medullary leucocythæmia, found that the xanthin bodies exceeded three to four times the normal amount. These changes in the urine are to be associated with those in the blood and organs described above, and probably all are due to the excessive breaking down of leucocytes, as it is now well established that xanthin and the lower members of the series are chiefly excreted in the more highly oxidised form of uric acid. Formic, lactic, and other organic acids in small quantities have been found in the urine in some cases; and peptone and albumoses have been observed occasionally. Albumin may be present towards the close of the disease, but, as a rule, the urine is free from it; hæmaturia, though occurring occasionally, is rare. There may be great enlargement of the kidneys due to leucocytic infiltration, without a trace of blood or albumin. Sulphates and phosphates have in some cases been found increased in amount, but this is not a well-recognised alteration. The occurrence of renal calculi from the increased excretion of uric acid and urates is not common, though a few cases have been recorded.

In the *skin* multiple tumour-like nodules, often reaching a hazelnut in size, have been recorded in a few cases. This condition was first described by Biesiadecki, and has been called by Kaposi "lymphodermia perniciosa." It has usually been associated with glandular enlargement, and the structure of the nodules has been described as resembling that of lymphoid tissue. Further observation appears necessary, however, to determine the exact relation of this change to leucocythæmia. A tendency to boils has been noted in some cases of leucocythæmia. Other changes in the skin have already been mentioned.

Symptoms in connection with the *nervous system*, apart from those produced by hæmorrhages, are on the whole rare. Mental affection, especially of a melancholic type, has been observed in some cases, chiefly towards the close of the disease; but it is not sufficiently frequent to indicate any special proclivity. In some of the acute cases delirium and coma have occurred before death, sometimes apart from marked pyrexia. In addition to the symptoms produced by cerebral hæmorrhage, which has been referred to above, paralyses of certain of the cranial nerves, due to hæmorrhage or leucocytic infiltration in their sheaths, have been recorded, and several observers have noted the occurrence of sudden deafness: in one or two cases this has been found to be due to hæmorrhage into the inner ear. In some other cases impairment of hearing, subjective aural sensations, giddiness, and the like, have been observed; and in one such case Politzer found a leucocytic infiltration of the structures of the labyrinth.

The *retina* on ophthalmoscopical examination very often shows distinct changes, which depend chiefly on the altered condition of the blood with the occurrence of hæmorrhages. When the anæmia is well marked the fundus is pale and sometimes of yellowish tint; the veins are usually dilated, tortuous, and paler than normal, whilst the arteries are narrow. There is sometimes swelling of the optic disc. Hæmorrhages in the retina are common, and are most frequently situated at the periphery, though they may also occur in the region of the macula. They vary in size, though they are usually small; in shape they are irregular and have sometimes a striated appearance. Pale spots, usually close to the vessels and often surrounded by traces of hæmorrhage, are also seen sometimes; occasionally they may reach a considerable size. They are composed chiefly of collections of leucocytes and degenerated nervous elements. In some other cases a uniform opacity of the retina has been observed, which has been found to be due to leucocytic infiltration of the layers of the retina. Interference with sight may be present or absent, according to the position of the lesions. As these are most common at the periphery, usually nothing abnormal is noticed by the patient, but in some cases, where the more central region is involved, defect of the field of vision may result. In a few cases such symptoms have first led the patient to seek advice, and in this way have led to the discovery of the disease. Hæmorrhage into the vitreous has already been mentioned as a rare occurrence.

Reproductive system.—In women there is often irregularity of the menstrual function. There is sometimes menorrhagia, occasionally metrorrhagia, but as the disease advances amenorrhœa is not infrequent. Women suffering from the disease have been known to pass through more than one pregnancy and to bear healthy children. In man the occurrence of persistent priapism, lasting sometimes as long as eight weeks, is a curious symptom which has been noted in a considerable number of cases. It has been attributed to thrombosis in the veins or in the sinuses of the corpora cavernosa, and in a case recorded by Kast evidence of such thrombosis was found after death, the priapism having occurred a year and a half before.

DIAGNOSIS.—In most cases of spleno-medullary leucocythæmia the diagnosis is very easy. Frequently attention is first drawn to the great enlargement of the spleen, and thereafter an examination of the blood reveals the nature of the disorder. The number of leucocytes may be so great as to leave no doubt possible; but it must be borne in mind that occasionally their number may not be much above normal, and also that in a number of other diseases the leucocytes may be increased in number. Here the characters of the leucocytes are of great importance. Such increase, known as *leucocytosis*, occurs in certain wasting diseases, in anæmia resulting from hæmorrhage, in acute suppurations, in various infective fevers, and so forth; and is no doubt produced by the circulation of certain abnormal products in the blood. Leucocytosis can be experimentally produced by the injection of many bacterial products, of peptone, nuclein, and other substances. The attempt to distinguish leucocythæmia from leucocytosis by the number of leucocytes is quite unscientific—the difference being one not merely of degree but of nature. In leucocytosis the increase is almost exclusively on the part of the leucocytes with multipartite nucleus, so that the proportion of these to the other leucocytes may be increased three- or fourfold, and no abnormal elements are present. In leucocythæmia, on the other hand, the leucocytes have the characters already described. As already stated, their number may fall in some cases nearly to normal, whilst the abnormal elements remain in the blood. Accordingly, when such a condition is found the case should be closely watched and the blood examined from time to time. Examination of the blood will also distinguish spleno-medullary leucocythæmia from other diseases with great splenic enlargement, such as ague or splenic anæmia. In the latter disease the number of leucocytes may be slightly increased, normal, or even diminished; but they never show the alterations in character met with in leucocythæmia.

In the lymphatic variety, that is, where the lymphocytes are in excess, the diagnosis is usually made easily in the same way. Such cases, with enlargement of glands, are sometimes mistaken for lymphadenoma, a disease of an essentially different nature. In the latter the glands are usually of firmer consistence, and often show matting; though this is not invariably the case. Of more importance is it that when the leucocytes are increased in lymphadenoma — their numbers sometimes reaching 25,000 per c.mm. or even more—the condition is a leucocytosis, and the cells have the character just described. Difficulty, however, sometimes arises in the case of children with enlarged lymphatic glands, in whose blood there may be a certain excess of lymphocytes, so that an early stage of lymphatic leucocythæmia may be suspected. In these cases examination of the blood from time to time will determine the matter.

Some cases of acute leucocythæmia with extensive hæmorrhages may be mistaken for severe purpura and like conditions; and this is the more liable to occur as the enlargement of the spleen may not be sufficiently great to attract special attention. In other acute cases, with high temperature and without special enlargement of lymphatic glands, the condition, as Ebstein points out, may even be mistaken for typhoid or other fevers. In such obscure cases the examination of the blood should always be undertaken, and will usually reveal the condition at once, if it be one of acute leucocythæmia. Here again the importance of distinguishing it from a mere leucocytosis may be noted.

Cases of disease sometimes occur in which diffuse leucocytic infiltrations of certain tissues—for example, of the intestinal mucous membrane—are present, which changes can scarcely be distinguished histologically from those met with in the lymphatic form of leucocythæmia, but are unattended by the characteristic change in the blood. It is quite probable that when the cause of such changes becomes fully known, it will be found to be the same in the two series, and some general term may include them both. In other words, there may be cases of the same disease, in some of which the lymphocytes of the blood are increased, in others not; just as in some cases of the lymphatic form of leucocythæmia the kidneys are sometimes affected, sometimes not. But so far as our present knowledge carries us, it is advisable to consider the blood changes as constituting the distinctive feature of leucocythæmia, and as forming the means of diagnosis.

PROGNOSIS.—Though we cannot affirm that leucocythæmia always ends fatally, yet, so far as prognosis is concerned, it must be regarded as a condition of the gravest nature. A few cases are recorded in which a cure is said to have taken place; but in most of these one cannot but regard the evidence as inconclusive, as the diagnosis in some of the cases was uncertain, and in others the subsequent history was insufficient. Cases, however, certainly occur in which great improvement in the general health takes place, the number of leucocytes also diminishing greatly; and this improvement may last for a year or two. Accordingly, while the disease practically always ends in death, the duration of life after the recognition of it is very variable. In some chronic cases the disease has lasted as long as seven years; in other cases it has run an acute course in a few weeks or less. In relation to the probable duration in different cases a few general facts may be given.

In the first place, as regards age, the disease is usually of shorter duration in young subjects, especially when it is of the lymphatic variety. The spleno-medullary form in adults, when there are no bad symptoms, is usually chronic, and often lasts one or two years. Some writers consider that it is rather more rapid in women, but there is probably little or no difference between the sexes in this respect.

The number of leucocytes in itself does not give much indication, though a progressive increase is an unfavourable sign. The degree of anæmia present is of more importance, and a steady decrease in the number of red corpuscles is especially grave. The size of the spleen affords little assistance, except, perhaps, that a very great enlargement points to a comparatively slow course so far, a circumstance which may sometimes affect the prognosis.

Enlargement of the lymphatic glands, when at all marked, is, as a rule, a bad sign, since it usually occurs either late in the disease or in cases which run a rapid course.

Hæmorrhages have a varying significance according to their position and extent. Hæmorrhage from the nose is not infrequent in the early stages of the disease, and, though it may lead to a fatal result, may occur from time to time in cases which run a very chronic course. Hæmorrhages from the stomach or bowels are much more serious symptoms, and usually indicate a condition of special gravity. So also hæmorrhages in the skin are generally the omen of rapidly advancing cachexia. The presence of dropsy, well-marked or continuous pyrexia, or persistent diarrhœa naturally makes the prognosis specially grave.

A judgment as to the course of the disease will be materially aided by observation for a time of the case under treatment. But it must not be overlooked that a patient suffering from leucocythæmia is in such a state that a complication or sudden aggravation may occur at any time, and prove fatal. Special attention has already been drawn to the incidence of cerebral hæmorrhage.

TREATMENT.—Leucocythæmia is a disease for which there is no specific remedy, and it is one which too often runs a steady course towards a fatal termination. But while this is so, under careful and judicious treatment life may be considerably prolonged in many cases, and great improvement may be effected in some. It ought to be regarded as a disease in which death may be much hastened by indiscretion on the part of the patient; but an intelligent knowledge of the features of the disease and the complications which are likely to arise will sufficiently guide the physician in this matter.

It is rather the rule than otherwise for patients in the earlier stages of chronic leucocythæmia to improve when under treatment in hospital. The regulation of the condition of the alimentary canal is of great importance. The diet ought to be arranged so as to exclude anything likely to lead to gastric disturbance, but otherwise should be as full and nourishing as the condition of the patient will allow. If a tendency to constipation be present, the bowels ought to be kept regular by mild laxatives or intestinal stimulants; constipation is apt sometimes to be followed by diarrhœa. Powerful purgatives, however, are contra-indicated in all conditions which may arise in the course of the disease. Excess in eating and drinking, exposure to cold, over-exertion, and such like must be carefully avoided. Such general measures as these, along with good hygienic conditions, have a distinct effect on the general condition of health apart from treatment with drugs. The tendency to hæmorrhage should be kept in mind in connection with any surgical interference which may be incidentally called for in a patient suffering from leucocythæmia.

A large number of drugs have been employed in the treatment of the disease, and with regard to each it may be stated that whilst in some cases improvement or even cure is recorded, in the majority it has been found ultimately to fail. Of all the drugs employed I believe that arsenic is of the greatest value, and in many cases great improvement results from its use. It ought to be given at first in ordinary doses, to be gradually increased, and pushed as far as possible. Under its use the number of leucocytes may diminish greatly and may even fall to normal; the size of the spleen also may become considerably less, though sometimes it is little affected. Arsenic has also been administered subcutaneously and by direct injection into the spleen, but there are manifest objections to these methods, especially when the hæmorrhagic tendency is well marked. Some observers consider quinine in large doses to be of considerable service, but I look upon it as distinctly inferior to arsenic. Good results have been reported from the use of phosphorus in one or two cases, but the general experience is that it is of no value. In other cases improvement has followed the use of tonic medicines—cod-liver oil, iron, with or without quinine in small doses, and chalybeate waters such as those of Pyrmont or Schwalbach. In my experience, however, arsenic is the only drug which seems to have a distinct effect on the leucocythæmic condition.

On the view that the spleen is the primary seat of disease, a number of measures have been adopted to produce diminution of this organ. Such is the use of certain drugs—eucalyptus, quinine, and piperine (Mosler), the faradic or galvanic current applied over the organ, electropuncture, the cold douche to the splenic region, and so forth. All these measures, I believe, are without effect.

Excision of the spleen has been performed in a considerable number of cases, but almost invariably with a fatal result; it must be regarded as absolutely unjustifiable, and it is also, I believe, useless. Transfusion of blood has also been tried without any satisfactory result. Inhalations of oxygen have been administered in a considerable number of cases, sometimes alone, sometimes along with other remedies, especially arsenic. In the hands of some observers benefit has followed, chiefly in the early stages of the disease; but in many cases this treatment has entirely failed. The amount of oxygen employed has usually been about 30 litres daily, though sometimes as much as 100 litres have been used. Bone-marrow has been administered recently in this disease, but we cannot as yet speak definitely of its effects. There seems to be no scientific basis for this treatment, yet in a disease in which all known remedies may be without avail the method is worth a fair trial. The marrow may be administered either in the fresh condition or in the form of prepared tabloids.

The complications occurring in the course of the disease and most frequently calling for treatment are the hæmorrhages from various sources, the gastric and alimentary disturbances, and, in the later stages, the heart-weakness, dyspnœa, and dropsy. All these are to be met by the usual methods. In the more acute form of leucocythæmia arsenic should also be tried, but usually all remedies entirely fail, and the aid of the physician is limited to relief of the more distressing symptoms.

Robert Muir.

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the cellular tissue, the peritoneum, the pleura, or the pericardium; but comparatively little whether it be dependent on disease of the heart or of the kidneys, or neither. I have dwelt upon this fact in a paper to which I may venture to refer (4). From this I reproduce on the following page a series of estimations concerning dropsy fluids which I made as opportunity offered, and I append an abstract of the average qualities of these effusions in various places and with various disorders, including my own observations and those of others. Many of the figures upon which this abstract is based are taken from the table here reproduced; the rest may be found in the paper to which I have already alluded.

The first fact which strikes us is the uniformity of the mineral salts. In every place and from every cause the mineral salts of dropsy fluids present about the same proportion, which is about that in which they occur in the blood. By whatever process they traverse the vascular walls this is apparently the same in every place and with every disease. This looks like some unvarying physical action, like osmosis or dialysis. It is not so with the albumin, which varies much more with the location than with the disease. Œdema fluid, whatever the cause may be, contains only traces of it; when the cause is cardiac the albumin is greater by about one-half than when it is renal. Pleural and peritoneal effusions are always highly albuminous; sometimes the pleural more so than the peritoneal, sometimes the reverse. When due to heart disease the effusions contain more albumin than when due to kidney disease. Peritoneal effusions due to cirrhosis of the liver occupy an intermediate position with regard to albumin. The smaller amount of albumin in the effusions of kidney disease corresponds with the reduced amount of this substance in the blood under the same condition. With this allowance, it may be said that the effusions of dropsy in each place are so much the same in every disease as to suggest that they are produced at least by similar processes. As to the saline ingredients, these are probably the results of dialysis from the blood. The albumin as a colloid body cannot transpire by dialysis, but obtains its passage by pressure, by secretion, or by both together.

It will be of interest to refer to some of the physiological facts which bear upon the matter. In virtue of dialysis, crystalloid substances in solution traverse membranes until the liquid on one side is as fully charged as that on the other. Colloids, such as albumin, do not so traverse, if the pressure on each side is equal; but if the pressure be unequal, albuminous fluids, serum for example, pass with facility through a dead membrane or parchment paper until the pressure is equalised on the two sides. Physical laws, it is needless to insist, are as active within the living body as outside it, though within it there may be other or vital laws which modify or counteract. Dialysis and transmission by pressure are physical; secretion is vital. In experimenting with membranes and liquids with regard to osmosis, and processes of dropsy so far as they can be imitated outside the living body, the fact which comes out with the greatest prominence is the influence of pressure in moving

AVERAGE CONSTITUENTS of Dropsy Liquids in 100 parts. Various observers.

	Pericardium.			Pleura.			Peritoneum.			Cellular Tissue.		
	Sp. Gr.	Alb.	Mineral Salts.	Sp. Gr.	Alb.	Mineral Salts.	Sp. Gr.	Alb.	Mineral Salts.	Sp. Gr.	Alb.	Mineral Salts.
Diseases of the kidneys	1009	...	1·55 (?)	1011	1·48	·803	1010	1·09	·92	1009	·22	·92
Diabetes, after saline injection	...	much	·75	1016	2·10	·84	1013	1·38	·87	1010	·38	·89
Disease of the heart .	1018	3·08	...	1013	2·11	·784	1014	2·34	·79	1008	·35	·81
Disease of respiratory organs	...	...	...	1013	1·91	·826	1017	3·13	·84	1008	·23	·77
Cirrhosis of the liver .	...	1·06	...	...	...	...	1013	1·65	·83	...	...	...

The averages are obtained from the figures, my own and those of others, given in the paper referred to. The statements are mostly numerous. Occasionally, notably in the case of diabetes, there is only one case available.

S IN 100 PARTS.—W.

PLE

Appearance.	Sp. gr.	Alb.	Mineral Salts.		
			Sol.	Insol.	Total.
...	...	·056 opalescent)	·853	·021	·874
Clear, straw	1008·2				
Clear, colourless	1007·3				
Clear, straw	1018·5				
...	...	·125	·866	·025	·891
...	...	·210	·898	·020	·918
Nearly clear	1010·7	·095	·733	·020	·753
...	...	...	·769	·025	·794
Slightly turbid	1013·2	...	·803	·034	·837
...	...	·340	·860		
...	...	·385	·870	·023	·893
Highly sanguineous	1016·7				
...	...	·230	·830	·031	·861
...	...				
...	...	·33	·758	·030	·788
...	...	·29	·795	·025	·820
Straw, slightly sanguineous	1015·5	·324	·776	·022	·798
Dark straw	1012·2				
Slightly turbid	1012·3	·495	·814	·030	·844
Faintly blood-tinged	1012·8				
Clear, dark straw	1014·2	·235	·749	·024	·773
...	...				
...	...				
...	...				
...	...	1·39	·774	·03	·804

To face page 668.

albuminous fluids through membranes. If an albuminous liquid be placed at different levels on either side of a membranous septum, the liquid will quickly traverse in the direction of least pressure until an equilibrium is attained. It is true that if we put on either side of the septum liquids having opposite or very different chemical properties, acid and alkali, alcohol and water, etc., we may get osmosis which will be abundant against gravity and pressure; but such large osmosis has always been between liquids which have contrasted beyond what is possible between the inside and the outside of vessels concerned in the production of dropsy. Osmosis, as an agent in the production of dropsy, may be put aside as taking little part in it. Pressure and some process analogous to secretion by the outer wall of the vessel, the one physical, the other vital, are to be taken into question. The action of the physical agent is certain and obvious, that of the vital is hypothetical. The over-ruling influence of pressure in the transmission of fluids needs no insistence; it is the chief agent in the circulation, and is concerned in all that depends on the circulation, exudation, nutrition, and cell function.

Theoretically, as has been said, the accumulation of capillary exudation which constitutes dropsy may be due to excess of production or diminution of removal. There is only one source of increased exudation, that is the capillaries. Absorption may take place in theory either by way of the veins or the lymphatics, and obstruction or arrest in either must be had regard to in the causation of dropsy. But arrest in the lymphatics may for the present purpose be put aside. Stoppage in these vessels, as we know, may give rise to the glandular swellings which constitute elephantiasis, or may occasion chyluria. These are not dropsy, but are special results of a different kind. The failure of absorption which produces dropsy has to do only or chiefly with the blood-vessels, to which channels, therefore, our attention may be limited in considering the origin of dropsy. The capillaries may either put out too much or draw off too little, and here lies the whole question. In renal and chlorotic dropsy we see the results of excessive outpouring; in cardiac and hepatic dropsy, and in that of local venous obstruction, we have mainly the effects of insufficient withdrawal.

The simplest, or, perhaps, I should say, the least complicated form of dropsy is that which depends on venous obstruction, and is presumably due to an exaggeration of the intravascular pressure produced by mechanical means. Local dropsies in consequence of local obstructions may be considered first; they should be easy to understand, and may help to throw light on less simple conditions. The incidence of local dropsy must be considered both in the light of experiments on the inferior animals and in regard to human pathology. Experiments made by disease on the human subject are entitled, if not to the first, yet to the greatest consideration where the human body is concerned. It was observed by Lower 200 years ago that ligature of the ascending cava in the dog caused œdema of the hind legs. Ranvier on repeating Lower's experiment failed to obtain the results until the section of the sciatic nerve was superadded, upon which œdema appeared on the side of the section, not on the other. It was shown, however, by Cohnheim that if the veins of the limb were obstructed by an injection of plaster of Paris, œdema presented itself though the nerves remained intact. Thus it would seem that venous obstruction in the dog, unless it be complete and extensive, does not suffice to cause exudation unless the blood-pressure is enhanced by vasomotor paralysis. In the human subject venous stasis by itself appears to be generally sufficient to cause this result. The dropsical exudation has to do only with the blood-vessels; the lymphatics which are not included in the obstructing process have no concern in it. The venous obstruction probably promotes dropsy in two ways: by increasing intracapillary pressure it increases capillary exudation; by arresting the venous return it hinders absorption so far as this process depends on the blood-vessels.

But there are other factors which complicate even this simplest form of dropsy. Nature provides many compensations and adjustments. A vein may be obstructed in a dog, or even in a man, and no dropsy ensue. Collateral circulation may keep down the pressure below the point which is necessary to give rise to dropsy. In otherwise good health dropsy is less likely to be developed than when certain abnormal conditions, other than mechanical, are superadded. Thus it has been shown by experiment that local dropsy is encouraged by depletion, or in other words by hydræmia. Watery blood yields the transudation more readily than normal blood. Ligature of the femoral vein in a dog may cause no œdema. But if the animal be repeatedly bled before the operation then œdema will follow upon it. Certain changes in the nutrition of the vessel walls, also, may allow of œdema which would not present itself were these normal. There is reason to believe that continued venous repletion and lack of proper circulation may render the capillaries leaky or more than normally permeable. Thus after ligature of a vein in an animal dropsy may not present itself at once, but only after the lapse of a time wherein the capillary walls have presumably suffered in their nutrition by reason of want of oxidation, or otherwise.

The clinical aspects of the dropsies of local venous obstruction will be considered later, it being only necessary here to refer to their varieties so far as they throw light on their mode of production. Œdema of the lower extremity is well known to follow upon thrombosis of its veins, as in phlegmasia dolens, and when coagulation of blood in these vessels is produced by causes apart from the puerperal state. Œdema in the same region, but to a less extent, is produced without absolute obstruction by mere retardation of the venous circulation, such as occurs with varicose veins. The upper limb and the side of the face and neck become swollen when an aneurysm or growth presses upon the innominate vein, sufficiently to obstruct it; and a similar accident to the vena cava produces œdema of the upper or the lower part of the body, according to the position of the obstruction in the upper or lower cava. Hydrops ventriculi may ensue upon occlusion of the lateral sinus, and further illustrations of the same process are provided by the occurrence of ascites as a result of obstruction of the portal vein, whether in the trunk by way of thrombosis, or in its distribution in the liver from contracting fibrous tissue. Thus in the human body, at least, we see that chronic venous obstruction, be the mode what may, suffices to cause œdema without the intervention of any other recognisable or constant agent. Enhanced pressure in the capillaries thus produced appears to be the primary and efficient cause, though certain intermediaries are necessary to its operation. Dr. Lazarus Barlow has shown by experiment that vessels wherein the blood has been at a standstill permit of transudation more readily than vessels not thus circumstanced, so that under increase of venous pressure dropsy does not ensue at once, but only after a little time has elapsed so as presumably to allow the vessel walls to undergo changes which make them more permeable. This, however, does not militate against pressure as concerned with the dropsy process, though it shows that intermediate steps are necessary before it can be established.

To take now the dropsy of heart disease, as presumably the next in order of simplicity to that of local venous obstruction, it becomes apparent that, simple as it may at first seem, it raises many questions and presents for estimation many factors. It is probable that we do not yet know all about this form, or about any form of dropsy. We have to consider vital as well as physical agencies. In the first place, we have to consider how far cardiac dropsy is due to diminished absorption and how far to increased transudation. At first sight this form of dropsy would appear to be due to diminished absorption by way of the veins, for their channels are undoubtedly abnormally full, and present abnormal pressure; conditions which it would seem cannot but interfere with absorption by their means. But there are other modes and processes which somewhat complicate the matter. Beyond diminution of absorption dropsy results from increase of transudation, which may depend on increase of intracapillary pressure, changes in the capillary wall which render them abnormally pervious, and changes in the blood, notably hydræmia, which render it more apt to transude. Under cardiac failure, produced for example by injections into the pericardium, Dr. Starling has shown that arterial pressure is diminished and pressure in the systemic veins increased. He infers that under these circumstances pressure in the systemic capillaries and small veins is diminished. So far as we may be guided by these conclusions, we see reason for diminished venous absorption, not for increased transudation. But the territory of the capillaries is wide, and it may be that Dr. Starling pushes his conclusion too far. We may readily believe that under cardiac failure the capillaries in connection with the arteries are under low pressure and imperfectly filled, but at the same time the capillaries in connection with the veins may be over-full. If this be so, transudation may occur in one part of the capillary system, not in another. Thus we may have increased transudation in heart disease as well as diminished venous absorption. Wherever continued stagnation of blood has existed, the vessel walls, if we may judge by experiments, regard to the general question of dropsy, not keeping for the moment to that of renal origin, it is instructive to contrast these facts with another which is provided by the course of diabetes mellitus. In this disease œdema may occur though the urine is superabundant. I have known the legs to swell, and that without any ostensible cause beyond the diabetes, though the urine amounted to fifteen pints a day. Thus it appears that in the production of dropsy, whether albuminuric or diabetic, other conditions have to be reckoned with beside the quantity of the urine.

Any theory of renal dropsy to be satisfying must explain, or not be inconsistent with, the following facts:—In acute renal inflammation of the ordinary kind, where the urine is scanty but not suppressed, and the arterial tension increased but not to the uttermost, œdema is an early result. When the disease and the consequent increase of tension have been long continued and given rise to cardiac hypertrophy, then the dropsy lessens and may be entirely removed, as if the hypertrophy of the heart were antagonistic to it. When renal disease is of very slow and gradual development, as in many cases of the chronic granular kidney, and the heart allowed time to hypertrophy gradually with the increase of arterial tension, dropsy may be for long or altogether absent. We here have absence of dropsy with extreme arterial tension. Finally, however, if the patient survive to the last stage, comes a time when the hypertrophy of the heart fails of its purpose, or is deprived of its effect, by superadded dilatation and mitral insufficiency; and then dropsy may present itself for the first time, or reassert itself if it have been removed under the circumstances indicated. The dropsy now is of a complicated nature; mitral regurgitation has much to do with it, and it may often be regarded as more cardiac than renal.

Pursuing the inquiry from a different starting-point, we find with obstructive suppression, when the urine is totally absent, the tendency to death is by heart-failure, and the arterial tension, as declared by the pulse to the touch (for here sphygmometric observations are wanting, though much to be desired), is abnormally low. Here dropsy does not present itself, however much the vessels might be expected to relieve themselves by this discharge. A similar absence of dropsy, nearly or quite complete, together with extreme diminution of urine, is sometimes observed in cases of exceptionally intense nephritis. Here we have failure of heart and pulse, and of dropsy little or none.

So far it would appear for the production of renal dropsy there is required increase of arterial tension, or, in other words, increase of intracapillary pressure. Diminution of urine contributes to cause dropsy, but does not suffice alone to produce it. Hypertrophy of the heart, so long as it is not otherwise altered, tends to prevent the development of renal dropsy, or, if it has occurred, to remove it. This action of cardiac hypertrophy must be largely attributed to the expansile force or suction power of the left ventricle, which must be increased in proportion to the thickness of the walls. The suction thus established or increased must draw upon the pulmonary veins, clear the lungs, make way for the current in the systemic veins, and facilitate the drainage of the tissues so far as it is due to these vessels. With regard to acute renal dropsy, another factor is to be recognised in the pulmonary inflammation, commonly shown as broncho-pneumonia, which often, but not always, accompanies it. This, by impeding the return through the lungs, must assist the dropsy process. The increase of capillary pressure which has been inferred as attending acute renal dropsy is not extreme, and probably not unassisted in causing the result. It has been inferred with much likelihood that there is a simultaneous change in the capillary walls, in virtue of which they become abnormally permeable. This has been regarded as inflammatory (Cohnheim), but since the characteristic products of inflammation are wanting we may presume that the change does not attain to this condition. It has been already shown that the products of dropsy of every kind resemble each other enough to indicate that they are the results of similar processes, and that they all differ essentially from the products of inflammation.

The starting-point of acute or recent renal dropsy must be the toxic condition of the blood due to insufficient elimination by the kidneys. The capillaries, though not muscular, have been shown to be contractile, and it must be presumed that they offer abnormal resistance to the passage of blood which is abnormal in certain respects. Increased capillary resistance involves increase of arterial tension and of cardiac effort. With the increase of intracapillary pressure it has been inferred, and it cannot be doubted, that there is a change in the capillary walls which makes them unnaturally permeable, since the increase of capillary pressure, to judge by the arterial tension as revealed to the educated finger, is not in early cases extreme, and not enough alone to account for the effusion.

The œdema of chlorosis is evidently nearly allied to that of nephritis. In chlorosis we have a toxic state of the blood owing to a retention of what should be excreted, not from renal, but uterine failure; and with this there is, as I have elsewhere shown (4), exaggerated arterial tension, and probably a train of circumstances similar to that which I have indicated with regard to nephritis.

THE VARIOUS FORMS OF DROPSY CONSIDERED SEVERALLY

I shall divide the subject as appears most natural and convenient, having regard sometimes to the origin of the dropsy and sometimes to its location. I shall take local effusions first, which are generally, but not always, due to venous obstruction, then cardiac dropsy, then renal, and, lastly, such as depend on changes in the blood other than those due to disease of the kidneys.

Of LOCAL DROPSIES the first to be considered is **hydrops ventriculi or chronic hydrocephalus.**

In dealing with this affection it is necessary explicitly to exclude effusions which depend upon meningitis or any other form of inflammatory action. The complete or almost complete absence of albumin in the fluid may be taken in assurance of its origin otherwise than by inflammation. The fluid of true hydrops ventriculi is but a superabundance of the natural cerebro-spinal effusion. It must be acknowledged at starting that all the causes of this excess are not understood, and that some of the causes to which it has been attributed are, to say the least, extremely doubtful. Venous obstruction, particularly in the lateral sinuses, appears to be an unquestionable, though not a frequent cause of intracranial dropsy. This result has been known to come upon obstruction of the lateral sinus by growths and cysts, though I cannot learn that it has been traced to thrombosis. This condition commonly produces acute changes in the brain which do not comprise dropsy; dropsy is usually a chronic process. As an example, I may mention a case which I have elsewhere given in detail (5), that of a child whose lateral ventricles were found to contain 8½ ounces of clear fluid, apparently as the result of the total obstruction of the right lateral sinus by a mass of tubercle which belonged to the cerebellum. There was no tubercle in connection with the brain with this exception, nor anything to account for the effusion beyond the obstruction of the sinus. Dr. Murray, of Newcastle, published a case in which a similar result was produced by a cyst of the cerebellum which compressed the veins returning from the lateral ventricles.

The frequent association of chronic hydrocephalus with rickets points to another mode in which it is probable that the intraventricular dropsy may be produced. The cerebro-spinal fluid, an excess of which constitutes the disease, is controlled in amount by the pressure against which it is secreted. The purpose, or at least the effect, of the fluid readily poured out and as readily reabsorbed, is to maintain a slight and uniform pressure on the nervous centres. The pressure is secured by the closure and indistensible character of the intracranial and intraspinal cavities. If the intracranial cavity be laid open so that its contents can freely escape, the controlling pressure is taken off, and the secretion proceeds without hindrance and with abnormal profusion. Thus, when the base of the skull has been fractured so as to make a communication between the subarachnoid cavity and the external auditory meatus several pints of the cerebro-spinal fluid have been known to escape from that exit in twenty-four hours. On the same principle, it may be presumed that if by rickets or other cause the cohesion of the cranial walls is impaired, they may yield to the pressure which the cerebro-spinal fluid normally exerts, and become expanded into the hydrocephalic state. But, however this process may account for certain cases of intracranial dropsy, it obviously has its limitations. Hydrocephalus may be intrauterine and a cause of difficult labour. For this other causes must be sought. Within the uterus the skull must always be exposed to considerable external pressure, and the dropsical accumulation cannot be attributed to the want of it.

The late Mr. Hilton propounded, in his book on *Rest and Pain*, a hypothesis of hydrocephalus which must be considered. The fourth ventricle, with which the other ventricles communicate, is partitioned from the subarachnoid cavity by a fold of pia mater which crosses the lower end of this ventricle. This, however, does not completely divide the ventricular from the subarachnoid cavity, for it is perforated by a small hole, commonly about the size of a pin's head, called the foramen of Majendie, which makes a communication between the two. In some cases of intracranial dropsy, but by no means all, this opening has been found to have been closed by inflammatory adhesions. Mr. Hilton supposed that the accumulation in the ventricles was due to the closure of this opening.[1] If this be so, we must suppose the fluid to be secreted within the ventricles and absorbed outside them. Thus the functions of the ventricular and subarachnoid cavities would be opposite; the ventricular secreting, the subarachnoid absorbing. In this view the function of the choroid plexuses would be only to secrete, that of the spinal cavities only to absorb. But we have no knowledge to warrant this assumption. The probability is that all the cavities under ordinary conditions both secrete and absorb, so that cutting off one cavity from another would not necessarily give rise to dropsical accumulation. Supposing the intraventricular secretion to be exaggerated by inflammation, or some action akin to it, the arrest of escape might then lead to accumulation, but the results of inflammation are not within my present scope.

It is not necessary to include in this bare outline of intracranial dropsy any clinical description of chronic hydrocephalus or any detailed consideration of the treatment proper to it, since these will be found in another part of this work. It may be briefly said that this condition is not one which yields readily to treatment, while some methods which have been employed are not free from danger. The disease in some instances is exceedingly chronic, and indeed may exist for a large proportion of the ordinary term of life. At an early period the treatment for rickets is often indicated. I have known a limited reduction in the size of the head to be produced by external pressure by means of a broad elastic band placed horizontally round the head, with the pressure so moderated as to cause no injury to the integuments. I have known the neglect of this moderation to be followed by sloughing and death. Judicious pressure before the skull is finally ossified may generally be expected to reduce the circumference of the head by from one to two inches. Evacuants, mercury, squills, digitalis, and iodide of potassium have been administered, but mostly with little beneficial result. Tapping has been employed, repeated, and survived; but this is not without its dangers, and appears to be seldom productive of tangible good. Even though no harm result the fluid usually reaccumulates. It must be borne in mind that the hydrocephalic head does not always increase in the same ratio as the body. Thus in the process of growth the head, though becoming absolutely larger, will become relatively smaller, and the disproportion between the head and the trunk may spontaneously lessen.

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[1] I have discussed the anatomical relations of the intra- and extra-ventricular cavities in the *Lancet* for 16th July 1870.

Other local dropsies need only brief mention here, since most of them will find more ample notice elsewhere. Perhaps the most frequent is that which ensues upon coagulation in the veins of the lower extremities, and is generally consequent upon changes in the blood, which may be septic, puerperal (probably generally septic), chlorotic or gouty. It was formerly supposed that the puerperal thrombosis, known as phlegmasia dolens, was essentially due to an inflammatory condition of the lining of the veins of the legs, which travelled after the manner of an erysipelas from the veins of the uterus to those of the lower limbs by anastomoses, which the elder pathologists were careful to display by dissection. In this view the plugging of the veins was the result of phlebitis, not its cause, as we now believe. The symptoms and treatment of phlegmasia dolens and other varieties of thrombosis will find a place elsewhere. It will suffice to say that this disorder, like many others, furnishes an example of local dropsy, due simply to venous obstruction, as are many kinds of dropsy which are met with in the course of human pathology, however this cause presents itself as qualified and complicated in experiments on animals. It is obvious that in the treatment of phlegmasia dolens the main object must be the safe removal of the coagulum ; in other words, its solution, rather than, as in old days, the reduction of the phlebitis by "antiphlogistic" measures. With our present knowledge, the best means of doing this appears to be the administration of alkalies, so as to keep the urine alkaline, and thus charge the blood to overflowing with the alkaline solvent. A mixture of bicarbonate and citrate of potash answers the purpose, to which ammonia and quinine may be superadded, in order to obviate any depressing effect which the potash salts might produce.

Among other local dropsies due to venous obstruction may be mentioned those which result from aneurysms and other tumours which press upon the veins from the outside, whether of the upper or lower extremities, and thus hinder the return of blood, and give rise to limited œdema. Such local dropsies are often important diagnostically as indications of venous obstruction.

The œdema of the legs of pregnancy, generally due to the pressure of the gravid uterus on the ascending cava, needs but a passing mention. When uncomplicated with œdema of the face or albuminuria it may be regarded as purely local and mechanical, and will pass off with the condition in which it has originated. This limited and mechanical œdema is apt to become complicated with renal disease, which will be indicated by albumin in the urine, by œdema elsewhere than in the parts drained by the inferior cava, and possibly by uræmic symptoms ; but I need not now dilate upon what has been fully dealt with elsewhere (see vol. iv. p. 380).

Ascites may be a limited or local dropsy due to portal obstruction, or part of a general dropsy of which the causes are manifold. It is to be considered now only as local and isolated. Hepatic ascites furnishes one of the most striking examples of dropsical effusion from venous obstruction. This occurs in the portal system, and may be either in the trunk of the portal vein by thrombosis, or in its minute distribution as the result of cirrhosis.

Portal thrombosis is a cause of ascites of ideal simplicity, but of no great constancy. To produce this result the thrombosis must be extensive, and the survival of the patient must be such as to allow it to become chronic. I cannot learn that portal thrombosis causes dropsy in its acute or recent stage, whatever may ensue after lapse of time. I have known death by syncope or collapse to be due to complete obstruction of the portal vein by embolism or thrombosis, and no sign of dropsy to be discoverable after death. It has been shown by experiments on animals that on ligature of a vein dropsy does not at once show itself, but only after a time; an interval being apparently required to allow of changes in the wall of the vessel whereby transudation is facilitated. So it would seem, in the human subject, that extensive or even total obstruction of the portal vein by clot may occur and cause death without any trace of dropsy. On the other hand, cases present themselves in which thrombosis is productive of much, even extreme ascites. I call to mind the case of a gentleman, the subject of cystinuria, who had ascites nearly to bursting, which was attributed to this cause—a diagnosis which was confirmed by his ultimate recovery. Many cases have been recorded, and some have come within my own experience, in which portal thrombosis has been testified to after death in association with ascites. I published such an instance in the 14th volume of the *Pathological Transactions.* In this the plugging was of old standing, and was such as to occlude the portal vein completely. The patient was a woman of twenty-one years of age. The liver was shrunk—it weighed only twenty-eight ounces; it was not markedly cirrhotic, and it was supposed that the atrophy was the result of the obstruction, not the cause of it. It is worth noting in connection with serous effusion of this origin that hæmorrhage in the portal territory, into the stomach or bowels, is a not infrequent result of obstruction of the portal vein by coagulum, as of obstruction of the same vein by other means.

Apart from thrombi the obstructions of the vein which cause ascites are several, the chief of which is cirrhosis; which indeed is by far the most frequent of all the causes of dropsy limited to the peritoneal cavity. Before proceeding to this, some other causes of portal obstruction and consequent dropsy may be briefly dismissed. One of these is malignant growth which may press upon the portal vein, and even has been known to intrude into it; this, like other causes of portal obstruction, may be a cause of hæmatemesis as well as of ascites. Syphiloma is another cause of hepatic ascites by way of gumma, cicatricial contraction, and fibrotic change. An infrequent and even a doubtful cause of hepatic ascites is lardaceous disease. It is a matter of common experience that general dropsy, including ascites, often ensues upon lardaceous disease which affects several organs. It is also well known that the liver may suffer the extremity of lardaceous disease, and yet no peritoneal dropsy supervene. It is certain that lardaceous disease of the liver has of itself little tendency to cause ascites.

With regard to cirrhosis, this is a cause of dropsy of which it is not easy to exaggerate the importance. In this disease the fibrous tissue of the liver becomes the seat of hypertrophy, new growth, and subsequent contraction, to the strangulation of the vessels which it surrounds. The new fibroid tissue, often highly nuclear, of the embryonic type, collects chiefly in the portal canals, and involves more particularly the minute ramifications of the portal vein; but other vessels of the liver, the hepatic vein as well as the portal, are affected in the same manner. By the strangulation, chiefly of the minute portal vessels, the portal circulation is retarded, and the blood caused to accumulate at high pressure in the venous radicles in which the portal circulation takes its origin. This leads to congestion of the spleen, often to hæmorrhage from the mucous membrane of the stomach and intestines, and to the exudation of serous fluid from the visceral peritoneum into the peritoneal cavity. Hence ascites is one of the common consequences of cirrhosis of the liver. Whether cirrhosis be attended with increase or diminution of the size of the liver, ascites may occur, but it is most likely to do so when the atrophic process prevails over the hypertrophic. The smaller the liver the greater is the tendency to ascites. But this is by no means a necessary consequence of cirrhosis of any kind; this change in all its varieties is frequently found after death without any such result. Cirrhosis has no direct tendency to cause œdema, though the lower extremities often swell under the influence of hepatic ascites as a secondary result of abdominal pressure. Another result of the pressure on the vena cava due to ascites is vicarious distension of the abdominal veins. A smaller degree of distension may be due to cirrhosis independently of ascites brought about by the various anastomoses which connect the portal with the systemic veins.

It is not necessary here to recapitulate in detail the symptoms of hepatic ascites; no form of peritoneal dropsy attains so great a degree; it is not unknown for the abdominal wall to give way at the umbilicus with discharge of the contained fluid.

The treatment of ascites will be considered in connection with that of dropsy in general.

Omitting hydrocele, though it might be placed in the category of local dropsies, I now proceed to the consideration of—

GENERAL DROPSY, or of dropsy which depends on causes acting on the system at large. Before proceeding to particulars I will introduce some details which bear upon the frequency of general dropsy as the result of the several causes to which it is due.

CAUSES OF GENERAL DROPSY, as revealed after Death in 300 Cases taken consecutively from the Post-mortem Books of St. George's Hospital, from the Year 1888 to the Year 1897.[1]

Causes of Dropsy.	Number of Cases.
Valvular disease of the heart sole or chief cause	136
Valvular disease and kidney disease not obviously consequent upon it	12
(Of the foregoing cases mitral stenosis present in	62)
(,, ,, ,, pericardial adhesion or pericarditis in	16)
Pericardial adhesions, valves healthy	2
Heart dilated or fatty, valves healthy	12
Aneurysm of arch, with or without valvular disease	11
Congenital disease of heart, with or without valvular disease	2
Disease of lungs or respiratory organs	19
Thrombosis, pulmonary and scattered	1
Kidney disease sole or chief cause	69
(Including large white, not lardaceous, and nephritis	20)
(,, granular and white contracted	43)
(,, destruction of one by stone or tubercle with consecutive disease of the other	6)
Lardaceous disease	17
Diabetes	2
Anæmia	2
Leucocythæmia	1
Sclerema	1
Tumours central or scattered, lymphadenoma, sarcoma, carcinoma, etc.	8
Cause uncertain, associated with tuberculous meningitis, tubercular peritonitis, pyæmia, dysentery, etc.	4
	300

The foregoing table giving the causes, as far as could be ascertained after death, of general dropsy in 300 cases, was the yield of 3185 necropsies, so that this condition is evident after death in about 1 in 10 of those who die in St. George's Hospital. It must be taken into account that small amounts of œdema which were apparent before death are sometimes not observable, or not observed, afterwards, so that the proportion of dropsy in life would be slightly greater than is recorded after death. It must also be borne in mind that the table indicates only dropsy, dependent on causes acting generally, to the exclusion of hepatic dropsy and that dependent on local causes. Had all varieties of dropsy been included, and this table based on observations made before death instead of afterwards, it is obvious that the proportion of dropsy to deaths would have been considerably greater than in the present estimation. Œdema, for which there was no local cause, has been accepted as an indication of general dropsy; in most cases there was also effusion in the serous cavities. In a few instances, where multiple serous effusions occurred without œdema, these were similarly regarded.

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[1] All forms of dropsy which are properly local rather than general are excluded; such are all the limited forms of œdema which depend upon obstruction of the veins of limbs. and hepatic dropsy which depends on obstruction of the portal vein. On this ground hepatic ascites is excluded, even though associated with œdema of the lower extremities.

Of the 300 cases the dropsy was due to affections of the heart or aorta in 163; to heart disease, together with kidney disease, neither, as far as could be judged, consequent on the other, in 12. Dropsy presents itself as a cardiac result nearly twice as often as of renal origin, even though all the lardaceous cases be reckoned as renal, which may properly be done. Of the cardiac conditions which give rise to dropsy mitral stenosis is by far the most frequent. Next to the heart in order of frequency come the kidneys as a cause of dropsy. Of the renal causes, though the large white kidney is more constantly thus followed than any other, yet the granular or contracted kidney, from its more numerous occurrence, more numerously gives rise to this symptom. Disease of the lungs and bronchial tubes takes the third place as a cause of general dropsy. Pulmonary dropsy, if not so common an event as might be expected from the position of the lungs in relation to venous return, is yet an occurrence of considerable frequency. In 19 of the 300 the dropsy was due to disease of the organs of respiration.

CONDITIONS of the Organs of Respiration to which Dropsy was apparently due in 19 Cases.

Emphysema alone	2
,, + bronchitis	4
,, + bronchitis and fibrosis of lung	1
,, + broncho-pneumonia	1
Phthisis alone	3
,, + fibrosis of lung	1
,, + bronchiectasis	2
Bronchiectasis alone	2
Fibrosis alone	1
Extensive pleural adhesions with obstruction of pulmonary artery by clot	1
Carcinoma, obstructing bronchus	1
	19

It may be of interest to look into the particulars. Of the pulmonary causes of dropsy emphysema takes the first place, being present in 8 of the number. This was notably associated with bronchitis in 4 cases, and, as this condition is less conspicuous after death than during life, it is likely that it existed in all. A fibrotic change in the lung was recognised in 7 cases. In this enumeration I have assumed, as may safely be done, that fibrosis was present in every case described as bronchiectasis. Phthisis, mostly advanced and attended with excavation and often combined with fibrosis or bronchiectasis, was present in 6. Thus emphysema and fibroid change are the leading factors of pulmonary dropsy, which may be explained by the effect which both these changes have in obliterating or removing the pulmonary vessels.

It has been shown that heart disease as a cause of dropsy outnumbers kidney disease by about 2 to 1; it may be worth while to ask which form of dropsy is the more severe, and which tends most to invade the serous cavities. I find that among the cases under consideration extreme or great œdema was relatively more common with renal than with cardiac disease. One in 2·2 of the renal cases, not lardaceous, had much or extreme œdema; 1 in 3·1 of the cardiac cases were thus affected. Of the lardaceous cases 1 in 2·8 had much œdema. As to dropsy of the serous cavities, 1 in 3·1 of the renal, not lardaceous, cases had double hydrothorax, or considerable effusion in both pleuræ, while 1 in 3·4 of the cardiac cases was thus affected. No instance of double hydrothorax occurred in connection with lardaceous disease. With regard to ascites, 1 in 2·2 of the renal, not lardaceous, cases presented this result; 1 in 2·5 of the cardiac. Of the lardaceous cases, 8 of the 17, or 1 in 2·1, had ascites. According to this showing, not only is œdema more extreme under renal than cardiac disease, but the serous cavities in renal disease are the more apt to participate.

That œdema should attain greater proportions in renal than in cardiac disease is what would be generally expected, but not so the preponderance of hydrothorax and ascites. But it must be considered that the foregoing enumeration is based on fatal cases, cases in which the utmost limit of time has been allowed for the development of complications. Cases essentially renal are more prone to acquire potential cardiac change than are cases primarily cardiac to acquire potential or vitally important renal mischief. Most of the renal cases before the end have become so complicated with consequent changes in the heart that both heart and kidneys may be looked upon as taking part in the result. Be this as it may, it would seem that advanced renal disease is productive of more dropsy within and without than advanced heart disease.

The obvious distinctions between cardiac and renal dropsy, or, to put it more generally, between dropsy of mechanical origin and that which has its origin in the state of the blood, may be briefly expressed. Renal dropsy is due to increased exudation, cardiac dropsy in chief to diminished absorption. Cardiac dropsy usually begins about the feet, and may affect other parts of the body before it reaches the face; renal dropsy is usually first apparent in the face, though the lower extremities soon participate. The dropsy of lardaceous disease resembles that of cardiac origin, insomuch that it begins in the ankles and is often long confined to the lower extremities. With regard to the great serous cavities, large dropsical effusions, whether into the pleuræ or peritoneum, are, as has been seen, common to renal and cardiac disease. It may be said, however, that these cavities are especially subject to dropsical invasion in connection with the large white kidney of nephritis and the advanced granular kidney in which secondary cardiac change has been superadded to the renal. Whether cardiac or renal, such effusions are amenable to treatment. With regard to renal dropsy, this has a tendency to go away of itself; the changes in the heart in connection with renal disease promote the removal of the effusion.

I will now revert to the several kinds of general dropsy, and give to each some separate consideration, and I will take cardiac dropsy first.

Cardiac dropsy and that dependent on pulmonary obstruction are essentially mechanical, and their immediate cause repletion of the veins, which increases intracapillary pressure and exudation, and diminishes absorption so far as this is due to the blood-vessels. The state of the heart and lungs is such as to obstruct the emptying of the systemic veins. The essential hindrance may hypothetically, and indeed actually, be placed where these vessels enter the right auricle. Here is the key, or rather the keyhole, of cardiac dropsy; not that the primary lesion is here, for it is usually farther on in the course of the venous blood, but it is upon this point as the place of discharge common to the systemic veins that the obstruction must tell if universal or general dropsy is to be produced. The cavities and the intermediate pulmonary system are thus placed in the order of nearness to the venous outlet, the hindrance at which is the proximate cause of cardiac and pulmonary dropsy:—i. the right auricle; ii. the right ventricle; iii. the pulmonary circulation; iv. the left auricle; v. the left ventricle. The nearer an obstructive lesion is to the common venous exit the more immediate and the more marked might the resultant dropsy be expected to be. Were the cavities of the heart only passively concerned in the transmission of the blood this might hold good, but all are actively concerned, though in widely different degrees. The left ventricle is paramount. This brings the greatest force to bear in driving the blood onwards when the mechanism works correctly, and drives it back with the greatest force when there is any defect which permits of regurgitation. Thus the left side of the heart is the overruling agent whether in promoting or retarding the circulation, and its effect upon the venous outlet is practically greater than that of structures which lie nearer to it.

ANALYSIS of VALVULAR DISEASE in 143 P.-M. Cases of Cardiac Dropsy.[1]

Valvular Lesion.	How often present.
Mitral stenosis	68
Mitral dilatation or insufficiency	38
Mitral disease of uncertain effect	25
Aortic stenosis	12
Aortic regurgitation or insufficiency	27
Disease of aortic valve of uncertain effect	28
Tricuspid stenosis	5
Tricuspid dilatation or insufficiency	24
Tricuspid disease of uncertain effect	3
Stenosis of pulmonary orifice	0
Pulmonary regurgitation or insufficiency	1
Pulmonary valve disease of uncertain effect	1
General valve disease not further particularised	1

The chief cause of cardiac dropsy is imperfection of the mitral valve, and, above all, mitral stenosis. Among the 143 cases of cardiac dropsy referred to in the preceding table mitral stenosis was discovered in 68 cases, mitral dilatation or insufficiency in 38, mitral disease of uncertain effect in 25. Aortic disease, whether obstructive or regurgitant, presents itself with much less frequency, while the only lesions affecting the right valves to any important extent are such as permit of tricuspid regurgitation. Whether obstruction or regurgitation at the mitral orifice be the more productive of dropsy may permit of question; both are undoubtedly effective in this relation, but it is not easy to separate the two. Mitral stenosis is almost invariably attended, not only with obstruction, but with regurgitation; the so-called presystolic murmur which is characteristic of mitral stenosis is, as I maintain, regurgitant. If this be so, of which I have no doubt, we recognise with mitral stenosis both hindrance to the normal advancing current and abnormal retrogression. With this double difficulty dropsy is doubly invited. Both tend to the repletion of the auricle of the lungs and of the veins. Next in frequency as a cause of cardiac dropsy comes the aortic valve. Aortic regurgitation, as the preceding table shows, is more often associated with dropsy than aortic stenosis. This probably is rather because the regurgitant fault is the more frequent than because it has the greater tendency to cause the result. Aortic stenosis interposes a hindrance which must tell upon all the circulation behind it. So nearly complete sometimes is the closure, so great the obstruction, that not only is it obvious that the retardation must reach the sources of the systemic veins, but it is even a wonder that the circulation was not long ago brought to a standstill. Aortic regurgitation stands in a very different relation. So long as this is the only error the course of the blood during systole is clear; it is only during diastole that there is any embarrassment, and that not by solid impediment, but by the intrusion of an abnormal current into the ventricle. This must compete with the normal flow through the mitral opening, occasion delay or difficulty at this point, and some of the results which ensue upon mitral stenosis. But this interference is less effective as a cause of dropsy than mitral regurgitation, enforced as it is by the systole of the ventricle. As a matter of experience, aortic regurgitation, so long as it remains uncomplicated, is but a minor cause of dropsy. The defect may exist for many years, and in a marked degree, and yet no dropsy ensue.

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[1] This table is based on the same series of cases as that from which the table at p. 680 was compiled. It includes, however, 143 cases of valvular disease instead of only 136, for several instances have been included in which valvular disease, though sufficiently definite, was not the sole cause of the dropsy. Where more than one valvular lesion was present, the same case is noted under more than one heading.

To revert to mitral regurgitation. This, as is well known, is by itself an effective cause of dropsy, but it often occurs in this relation as a consequence and complication of other disorders. Mitral insufficiency may result from the dilatation of the ventricle and of the mitral orifice which ensues upon aortic regurgitation, and thus may determine dropsy, which the aortic fault alone might fail to produce. Again, extreme aortic stenosis may, as I have elsewhere shown (4), occasion mitral regurgitation by increasing the intraventricular pressure, and thus forcing a healthy mitral valve to leak. By this process any dropsy due to the aortic constriction could not fail to be enhanced. And outside the limits of disease primary to the heart, it may be said that mitral regurgitation is one of the causes of renal dropsy. I may have to touch upon this again, but I may briefly draw attention to the fact that late renal dropsy is often cardiac and mitral. The hypertrophy of the heart is succeeded by dilatation. This involves the mitral orifice and regurgitation ensues, though the valve may be unaffected except by stretching.

As to the right valves the only defects which present themselves with any frequency in relation to dropsy are, as the annexed table shows, such as permit of tricuspid regurgitation. Tricuspid stenosis sometimes occurs together with mitral stenosis, and from the same causes, but dilatation or insufficiency of the valve is much more common. This condition occurs more often in connection with changes in the ventricular wall, such as are due to mitral disease, than to disease originating in the valve itself. When present it cannot but be co-operative in the causation of dropsy, directing as it does a regurgitant current directly upon the venous exit. Though driven only by the comparatively weak right ventricle, it has every advantage of situation in its morbid action. Theoretically diseases of the right side of the heart bearing upon the systemic veins more immediately than those of the left side should have more to do with the causation of dropsy, but experience shows that this is far from being the case, and I have sufficiently indicated the reasons. To these may be added the fact that the left side, as more functionally active than the right, is more liable to disease.

Besides the valves, the state of the ventricular walls, especially of the left, has to do with dropsy. Expansion of this ventricle in diastole is an agent in carrying on the circulation not less real, though less potent, than its contraction in systole. Its expansion helps to empty the auricle, and thus indirectly draws upon the venous system by suction. The thinner the wall the weaker must be this action, while it must be proportionally increased by its hypertrophy. Thus attenuation and dilatation of the left ventricle must be placed among the conditions which contribute to cardiac dropsy, and, to anticipate what I shall have to revert to in connection with renal disease, hypertrophy of the left ventricle is the antagonist of dropsy, and may even be called Nature's remedy for it.

The lungs must not be disregarded as causes of a dropsy, which if not truly cardiac, is akin to it. I have already shown in detail, which I need not repeat (see page 681), that general dropsy is due in a certain minority of cases to causes which obstruct the pulmonary circulation, notably emphysema and fibrosis.

Renal dropsy.—Proceeding now from cardiac dropsy to renal, we pass from the more simple to the less. Renal dropsy and dropsy of other kinds also present an inverse relation to the amount of urine, which is general but not invariable. The urine may be superabundant, as in diabetes, and the legs œdematous, or the urine may be absent and dropsy absent too. With diabetes I have known, as I have said, the legs to become œdematous with the urine amounting to 15 pints a day; and with obstructive suppression dropsy may be as completely wanting as is the urine. As a rule with nephritis the urine is diminished and œdema present, but sometimes when this disease assumes an exceptionally acute form the urine may be reduced to almost nothing, and at the same time there may be almost nothing of serous effusion. Thus it appears that other factors have to do with dropsy, whether renal or diabetic, besides the discharge of water by the kidneys. Dropsy fluid, much the same in every disorder, though not in every place, is the capillary exudation, of which either too much is poured out or too little removed. In renal dropsy, to which our attention is now limited, the question narrows itself for the most part into excess of exudation. It is true, under certain circumstances which will be presently considered, renal dropsy may be enhanced by pulmonary embarrassment, which hinders venous return and probably hinders absorption; and it is equally manifest that hypertrophy of the left ventricle bears a part in removing renal dropsy by the increase of suction which it entails, and the consequent promotion of absorption by the veins; but in its most common forms, and while free from complications, renal dropsy may be regarded as the effect of increased outflow, and our attention may in the first place be directed to see how this is brought about.

Renal disease which is productive of dropsy, putting aside the lardaceous variety, is attended from the first with increase of arterial and cardiac tension, which increases as the disease goes on, and becomes attended with hypertrophy of the heart and arterial system. With the increase of tension dropsy comes, and continues together with it possibly to the end, or until the process of recovery gives a gradual finish both to the over-tension and its attendant exudation. But there is a later phase which all are not permitted to enter upon. Should time be granted and the disease assume a chronic form, then, with establishment of much arterial thickening, great ventricular hypertrophy, and further increase of intravascular tension, the dropsy may diminish, and even in the fulness of time be completely removed. Thus hypertrophy of the heart presents itself as the antagonist of dropsy, or at least as concurrent with its removal.

The complicated process which has been sketched in outline, partly morbid and partly remedial, requires to be considered in further detail. The hypertrophy is at once the result and the evidence of the over-tension. The tension must be attributed to the abnormal difficulty in the emptying of the heart and arteries. Then comes the question where the difficulty or obstruction lies. Wherever it is, it is attended with the increased capillary transudation which constitutes renal dropsy. The obstruction must be either in the arterioles or the capillaries. This question has been long and perhaps sufficiently debated. It is certain that the exudation is from the capillaries, and presumable that there is increase of blood-pressure in this situation. The arterioles are obviously thickened, with regard to the capillaries observations are wanting. But it is to be inferred, since the transudation from them is increased, that their blood-supply is not diminished, as it would be by any constrictive or stop-cock action on the part of the vessels which feed them. We may, therefore, regard the capillaries both as furnishing the source of the dropsy-fluid, and also of the initial resistance to which the subsequent cardio-vascular changes are due. We must presuppose an alteration in the blood, probably partly toxic and partly hydræmic, which causes abnormal resistance in the channels of these vessels, and occasions their walls to transude abnormally. It has been supposed that renal dropsy is akin to, or even equivalent to inflammation; that the outpouring of fluid is by a process essentially the same as that which constitutes inflammation. With inflammation there is certainly obstruction in the capillaries and exudation from them. With renal dropsy there is exudation from the capillaries, and presumably obstruction within them. But that the two processes are not the same is evident from the differences which exist between the products of inflammation and of dropsy. Inflammatory exudations differ from those of dropsy in their higher specific gravity, in their containing corpuscular elements in greater abundance, in their being more albuminous and more ready to coagulate spontaneously.

The late removal of renal dropsy on the establishment of cardiac hypertrophy cannot but be associated with, though, perhaps, it is not wholly to be attributed to, the suction action of the left ventricle, which must, unless dilatation intervene, be magnified in proportion to the thickness of the wall, so that an abnormal force is brought to bear upon the emptying of the auricle, and in necessary sequence upon the relief of the lungs and right side of the heart, and ultimately upon the clearing of the systemic veins. Another cause which must tend to the diminution of dropsy in the late stages of inflammatory disease of the kidney is the usual supervention of fibrotic changes in this organ, together with a further increase of vascular tension and increase of urine which before was scanty.

A point which must be adverted to in connection especially with acute renal dropsy is the state of the respiratory organs. Obstruction in these by emphysema or fibrosis has been noticed as a sufficient cause for dropsy in a chronic form, and it is probable that obstruction of other and more acute kinds may at least be co-operative in causing dropsy of corresponding acuteness. I have shown in detail, in the seventy-fifth volume of the *Medico-Chirurgical Transactions*, that in about two-thirds of the cases of acute renal dropsy the respiratory organs are the seat of some inflammatory process, often bronchitis or broncho-pneumonia. These conditions, though not the essential cause of the dropsy but probably only connected with it as the results of a common cause, cannot but enhance the dropsical tendency. With regard to pleural effusion, whether early or late, this is at once a result of the dropsical proclivity and a cause of its increase by the pulmonary obstruction which it occasions. These complications of renal dropsy are inflammatory and mostly acute. They are not such as produce any considerable hypertrophy of the right ventricle. This ventricle is comparatively little affected in renal disease. The contrast between the right ventricle and the left is one of the noticeable facts of Bright's disease. Both are hypertrophied, but the right to a slight and almost insignificant extent. The hypertrophy of this ventricle in renal disease is much less than in certain conditions of disease proper to the heart itself; whereas hypertrophy of the left ventricle is nearly as great under renal disease as from any cardiac lesion. In the paper to which I have referred I have given outlines of the ventricles in section which show among other things the relatively small hypertrophy of the right ventricle in renal disease.

These outlines may be appealed to as of interest with regard to the lardaceous disease, the relation of which to dropsy must next be briefly touched upon. With this disorder there is usually no hypertrophy of the heart and no increase of intravascular tension. The condition is obviously different from the dropsy connected with other states of the kidney. It is associated rather with want of force in the circulation than with exaggeration of it. The heart after death is often found to be somewhat dilated and enfeebled rather than strengthened. It is not improbable that, together with other factors, cardiac failure may have something to do with this form of dropsy. It is to be observed that the œdema of lardaceous disease often resembles that of cardiac origin in affecting the lower extremities in preference to the face.

Next to renal dropsy may be properly placed that of chlorosis as having points of resemblance to it. In making the inquiry to which I have already referred (4), with the aid of the sphygmograph, I came upon the fact that with the dropsy of chlorosis the tension of the pulse was increased, notwithstanding the pallor and general weakness of the patient. The resemblance which chlorotic bears to renal dropsy is evident. With both we have a toxic condition of blood due to the failure of an excreting organ—in one of the kidney, in the other of the uterus. The chlorotic state requires for its relief, as is well known, not only iron to obviate the anæmia, but remedies which stimulate the secretions of the uterus and bowels. The familiar mixture of iron and aloes owes its efficacy to its double action, at once depurative and restorative. In idiopathic or pernicious anæmia, extreme as the anæmia may be, we have no evidence of toxic retention, and as a rule there is no œdema; though the rule is not without exception.

Other forms of anæmia, or hydræmia—for the conditions, though not the same, are commonly associated—tend to produce or assist in the production of dropsical effusions. It is a tradition that in the old days of depletion patients were sometimes bled into dropsies. Marshall Hall, in his treatise on the *Effects of Loss of Blood*, includes a proneness to œdema and serous effusions, which result, he tells us, has long been remarked by medical writers. Practice at present is less illustrative in this respect. Dropsy from blood-letting, perhaps, does not admit of very exact statement, for we do not know how much to attribute to the disease for which the patient was bled, or to other disorders which the patient may have had, but which were not within the knowledge of the year 1830. Before taking leave of the relationship of anæmia to dropsy, I may revert to the obvious association of this state of blood with renal dropsy, especially of the acute kinds. Here anæmia is often extreme, and we cannot but suppose that it is contributory to the effusion. This connection is especially worth noting, as it is of practical importance.

Hydræmia may be a cause of dropsy or of its aggravation. Water can usually be drunk in large quantities without any effusion, providing that the glandular exits are free; the kidneys, bowels, and skin carry off the excess without any accumulation in the body, and in certain forms of renal disease, where the kidneys retain their power of response, good may result from this irrigation of the system; but when the kidneys are too far gone to answer to the appeal, enforced water-drinking may cause an immediate increase of dropsy. I once witnessed this in a case of renal ascites as the result of the administration of a pint of water every four hours. The tubular structure of the kidney was found to be extensively atrophied (3). As bearing upon the dropsy of hydræmia I may recur to a case of diabetic coma, in which 22 pints of a saline solution were injected into the veins in the course of 32 hours, with the result of slight œdema and effusion into the peritoneum, pleuræ and pericardium. Most of the effusions contained blood, whence it was inferred that intravascular pressure caused by the injection had much to do with the result.

The dropsy of diabetes is a paradox and a lesson. Great diuresis with this disease, instead of draining the tissues, may be accompanied with the accumulation of fluid in them. It may be paradoxically stated that the more that is taken out the more remains behind; while it may be instructive to consider a condition in which the amount of urine and of dropsy do not display the usual inverse relation. The passage of saccharine urine is often attended with renal irritation and nephritis, mostly tubal, but sometimes interstitial. This may give rise to dropsy which is truly renal; but, apart from secondary kidney disease, diabetic dropsy has long been recognised. Prout mentions "incurable dropsy" as one of the results of the disease, and tells us that at a late stage the urine diminishes, loses much of its saccharine property, and the feet and legs become œdematous. But this is not the whole story. The dropsy is not always incurable, nor does it always come on with diminution of urine. The legs may swell while the urine is still profuse. I have already alluded to such a case. The pulse was weak, without any trace of albuminuric tension. The dropsy disappeared with much promptitude under perchloride of iron, though it reappeared to some extent before death. I think it may be inferred that diabetic dropsy has its immediate origin in cardiac weakness and anæmia. It may be observed that the effusion affects the lower extremities rather than the face. Traces of pitting may often be detected about the tibiæ in this condition, though the patient may be much emaciated and the dropsy not obvious.

The treatment of dropsy is considered in connection with the several diseases of which it is a symptom, but a few general remarks may be here superadded, together with some brief reference to the principles which should guide our attempts to relieve its more important varieties. First may be placed the regulation of posture in reference to cardiac disease and to œdema of every kind. The lessening of venous pressure in the limbs, and the sparing of labour to the heart by the horizontal posture, are too obvious to need insistence. But with heart disease there is sometimes the difficulty that in consequence of the state of respiration the patient cannot endure to lie down, in which case the vital necessity must be first considered, and the dropsy in the second place. The disadvantageous posture of orthopnœa may in some sort be turned to advantage, for this attitude is favourable for the drainage of the legs after puncture. Not only with heart disease, but also with renal œdema is the horizontal position beneficial. Under this influence and the modification of blood-pressure which it entails, œdema of this origin will often disappear from the legs, and that without reappearing elsewhere.

Diet in dropsy may be considered before drugs. As dropsy fluid, whatever else it may contain, mainly consists of water, the question at once presents itself, What is the effect upon the accumulation of cutting off the supply? "If we indulge in harmless fluids we get the dropsy," was "a soothing reflection" of Mr. Pecksniff's, and one which, in certain conditions, as I have shown, is not altogether without warrant. How far does the converse hold; to what extent can we diminish the dropsy by diminishing the drink?

I have made experiments on the influence of dehydration by diet upon dropsy of various kinds,—cardiac, renal, hepatic, and ovarian; and in cases of large effusion the result of pleurisy. The daily drink has been reduced, in most cases gradually, to quantities of which the following may serve as examples. The daily quantities are expressed in fluid ounces—16, 15½, 14, 12, 11, 6, 4, 2. The liquids were generally tea or milk, generally both, sometimes with a small quantity, from three ounces to half an ounce, of gin or brandy. Rarely a lemon was given, which was reckoned as an ounce and a half of liquid. Acidulated drops were sometimes allowed. The foregoing statements include everything that was given in a liquid form; ordinary diet was generally allowed; the water contained in the solids is, of course, not included. The privation was generally well borne, better than I had expected; the tongue usually remained moist; if it became dry, the treatment was discontinued. Under this process of desiccation the appetite diminished, the patient usually lost weight, and generally, but not always, the dropsy notably diminished and sometimes disappeared. It was difficult not to suppose that in some of these cases the patient supplied himself with liquid at the expense of his accumulations. To mention renal dropsy first, I may say that I experimented on deprivation of water in this condition with extreme caution and apprehension, never pushing the restriction far or continuing it long. Œdema and ascites both lessened under the process, but the deprivation was ill borne, and I soon came to the conclusion that such experiments on renal dropsy were unjustifiable. With renal disease, such as tends to uræmia, an abundant supply of water is indicated to wash out the toxic products, and irrigation rather than desiccation called for.

With regard to dropsy depending on valvular disease of the heart, dry diet is generally harmless and sometimes beneficial. Of three cases in which this was employed without other treatment, in two the effusion, which was limited to œdema, was much reduced and the patients benefited. In another in which there was great ascites without œdema, the dry diet was conjoined with repeated tapping. After five operations in the course of three months and ten days, the first yielding 17 pints 12 ounces, the last 5 pints 18 ounces, the fluid ceased to re-collect, and the patient left the hospital without any. The dry diet was continued all the time; the daily liquid touched a minimum which was represented by 2 ounces of tea, 2 ounces of brandy, and 1 lemon. It is, of course, possible that the fluid might have ceased to reaccumulate had no treatment been employed, but I think this is scarcely likely.

Dropsy from cirrhosis of the liver is under most circumstances intractable. Of six cases treated by dehydration two only received decided benefit. One patient unsuccessfully treated lost a stone in weight in three weeks without any decrease of the ascites. Of the two successful cases one was that of a man with an enlarged liver presumed to be cirrhotic and ascites, for which he was tapped twice with reaccumulation. He then, the belly being distended, but not tightly, was put upon dry diet without medicine. After fourteen days of this no fluid could be detected. The regimen was continued for some time longer, and was ultimately discontinued without any reappearance of the fluid so long as he was under observation.

The other case was a somewhat remarkable one; it was that of a boy seven years of age, who had extreme ascites with hypertrophic cirrhosis and suspicion of alcohol. The enormous distension and the dyspnœa which it occasioned made tapping imperative, and between April 28 and June 21 this was done ten times. The dates and amounts were as follows: April 28, 80 oz.; April 29, 72 oz.; May 5, 89 oz.; May 10, 95 oz.; May 15, 117 oz.; May 26, 114 oz.; May 30, 126 oz.; June 7, 150 oz.; June 14, 150 oz.; June 21, 65 oz. The hopelessness of this continual tapping suggested to me treatment by dehydration. Accordingly, on June 29, when much fluid had again accumulated, and the belly attained at the umbilicus a circumference of 30 inches, dry diet was begun. No further tapping was required. The fluid diminished, disappeared, and never reappeared. On July 21 none could be detected, and the circumference had fallen to 25 inches. At its greatest reduction the drink was limited to 6 oz. of milk, 1½ oz. of brandy, and a few small pieces of ice. The restriction was well borne, and, so far as the ascites was concerned, the cure was complete. The restriction was continued in a modified way for some time after the loss of the ascites. The patient left the hospital practically well, and so remained for over two years, when he was brought back with an abscess in the brain, which apparently had nothing to do with his former ailment. This caused his death, and gave an opportunity for a post-mortem examination. The liver was contracted and markedly cirrhotic, fibrotic and hob-nailed. The peritoneal cavity contained no fluid, but was closed by delicate adhesions. It may be observed in retrospect that the cessation of the morbid secretion was not the result of the adhesion, but occurred prior to it. The belly was largely distended when the dropsy began to diminish. The apposed surfaces adhered after the cavity had been emptied and kept empty by dehydration; probably the precedent dropsy brought about a sub-inflammatory state of the membrane which invited adhesion when contact was established and maintained.

I may mention with brevity that in two instances of large effusion, the result of pleurisy, the fluid quickly disappeared under dry diet; this, of course, is not conclusive as to the effects of the treatment, for the fluid might have been absorbed had none been employed. A more striking example of the results of dehydration was afforded by a woman who had an enormous unilocular ovarian cyst simulating ascites. Under a diet which at its narrowest limitation comprised no liquid but what was contained in 2 oz. of tea and 1 oz. of brandy in the twenty-four hours, the patient lost in three weeks 3 inches in girth and 13¼ lbs. in weight. She ultimately underwent ovariotomy with success.

From the foregoing cases and other experience, I conclude that dehydration by diet may be used under certain circumstances in the treatment of dropsy other than renal. With cardiac dropsy, whatever its seat, this is generally harmless and may sometimes be useful. With regard to hepatic ascites, considering the safety of tapping and the immediate relief afforded by it, I think that dry diet may be best employed, not as a substitute for the operation, but as an adjunct to it, as in the case of the boy with cirrhosis, of which details have been given.

The routine remedies for dropsy can be only cursorily dealt with. The removal of fluid by agents which act on the secretions of the bowels, kidneys, and skin is familiar practice. Hydragogue purgatives, elaterium, compound jalap powder, sulphate of magnesia, and bitartrate of potash must receive acknowledgment, but are too well known to need insistence. With regard to renal dropsy, useful as such remedies sometimes are, they must be used with caution lest anæmia be promoted and dropsy thereby encouraged. It often answers well to mix a little saline purgative with iron in the periodic mixture. Neither should the diet be too parsimonious. But I need not repeat here what I have said in a previous volume (vol. iv. p. 401). Whether regarded as purgative or as acting otherwise, small occasional doses of calomel are often of use in renal as in cardiac dropsy, having regard to the intolerance of mercury in renal disease. Hot-air baths are often of especial use in renal dropsy, for they not only draw off the fluid, but relieve the blood of the impurities upon which the dropsy essentially depends. Of the remedies of the diuretic class those are most valuable which are also cardiac tonics, such as digitalis and squill. We know of no remedy which is of equal value in dropsy, whether cardiac or renal, to digitalis. In cardiac dropsy no combination serves as well as the time-honoured dropsy pill of Matthew Baillie—digitalis, squill, and mercury. Squill alone, or in other associations, is often disappointing. The same may be said of most other so-called diuretics. An exception as regards renal dropsy must be made in favour of the alkalising salts of potash, tartrate of potash, potassio-tartrate of soda, and citrate of potash; these are both purgative and diuretic, and if pushed to alkalinity of urine may do good by lessening the coagulation of fibrin in the form of casts. This extreme dosage is, however, but seldom called for. I must not omit to enforce the necessity of iron, and the avoidance of a very poor diet, in renal dropsy when this is associated with anæmia. The inutility of diuretics, and indeed of drugs generally, is especially apparent in ascites of cirrhotic origin.

The relief of dropsy by puncture may be touched upon. The tapping of the belly has already been adverted to. Its safety and utility are well known. It is better done with the aspirator or Southey's tubes than with the large trocar formerly in vogue. Relieving the abdomen also relieves the legs by facilitating the return by the vena cava. Where the pleuræ share in general dropsy I have often found it beneficial to the general condition to tap one or both of these cavities, which relieves the pulmonary circulation, and by consequence the general dropsical state. Though relief of the serous cavities indirectly relieves the œdema, the converse does not hold good. Draining the œdema does not relieve either pleural or peritoneal accumulation. But with its limited purpose it is often of use, though attended with more danger than the tapping of the serous cavities. Puncture of the legs is, however, a less formidable operation than it was before the invention of antiseptic surgery. The dangers which are to be apprehended are erysipelatous inflammation and cellulitis, which may suppurate and constitute the beginning of the end. Incisions should be avoided, and acupuncture or Southey's tubes employed; I have found the tubes on the whole to answer best. I have known enormous quantities of fluid to be drained off by these means. Two of these tubes in each leg in a case of renal dropsy drew off nine pints in two days, and I knew an instance of cardiac dropsy in which twenty-two pints were drained off as the result of a similar operation.

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HEART DISEASES

- CONGENITAL MALFORMATION OF THE HEART
- DISEASES OF THE PERICARDIUM
- DISORDERS OF FUNCTION, INCLUDING STRAIN
- INJURIES BY ELECTRIC CURRENTS OF HIGH PRESSURE
- DISEASES OF THE ENDOCARDIUM
- DISEASES OF THE MYOCARDIUM
- CHRONIC VALVULAR DISEASES—
  - DISEASE OF THE AORTIC AREA OF HEART
  - DISEASES OF MITRAL VALVE
  - RIGHT-SIDED VALVULAR DISEASES
- ANGINA PECTORIS

CONGENITAL MALFORMATION OF THE HEART

SYNOPSIS

SECTION I

Defects in the septa of the heart.
Stenosis and atresia of the pulmonary artery.
Stenosis and atresia of the aorta.
Transposition of the primary arterial trunks.
Premature closure or patency of the fœtal passages.
Irregularity in the number or form of the valves.
Anomalous septa.
Misplacements of the heart.
Deficiency of the pericardium.

SECTION II

(*Causation*)

Fœtal endocarditis.
Mal-development.
Development of normal heart.
Mode of formation of septal defects, stenosis and transposition.

SECTION III

Symptoms.
Cardiac signs.
Duration of life.
Causes of death.
Treatment.

THE subject of malformation of the human heart is one of great interest, and has attracted the attention of medical observers since the beginning of the century, but in more recent years these anomalies have been subjected to a thoroughly scientific investigation. The earliest observations consist for the most part of descriptions of morbid specimens, which are scattered through various periodical publications. From time to time these have been collected together, and have formed the subject of dissertations or lectures. One of the first of these was a dissertation by Meckel in 1802, a descriptive account drawing attention to the curious resemblance presented by some of the monstrosities to the hearts of reptiles, amphibians, and crustaceans. Chapters on the subject also appear in various works by Corvisart, Laennec, Hope, and others. A special essay by Dr. Farre in 1814, and a series of lectures by Dr. Norman Chevers in 1845 on *Morbid Conditions of the Pulmonary Artery*, drew particular attention to the very frequent anomalies of this vessel. In 1855 Dorsch insisted on the importance of fœtal endocarditis as a determining element in the causation of these abnormalities, a hypothesis which became too one-sided in its application.

Peacock, in 1855, was the first to issue a systematic treatise on the subject, a work which is stamped throughout with the most accurate observation. A new edition of the same work appeared in 1866, and in the preface

Peacock reminds us that it is but recently that attempts have been made to reduce the different forms of irregular development to any scientific arrangement, or to explain their nature and mode of production.

In the classification of malformations of the heart he is guided partly by the period at which the development of the organ becomes arrested or perverted; partly by the degree of impediment to the circulation which such deviation occasions, and the consequent interference with the functions of the heart after birth.

In 1875, at Vienna, Rokitansky published his most important monograph on the *Defects of the Septa of the Heart*, in which he differed from the current views of the development of the septa, and insisted on the importance of studying the anomalies in connection with the different stages of development.

In my treatment of the subject I have been guided mainly by the works of Rokitansky, of Peacock, and of Rauchfuss.

Section I. is devoted to a descriptive account of the commoner forms of malformation of the heart. In Section II. the mode of formation of the anomalies is explained as far as possible by reference to the processes of normal evolution. Section III. contains some of the more important phases in the life-history of the subjects of malformation.

SECTION I

SYNOPSIS.—*Defects in the septa of the heart—Complete absence of both auricular and ventricular septa—Defects in the auricular septum: Defect of the primary septum; Defect of the secondary septum; Patent foramen ovale—Defects in the ventricular septum: Complete defect; Defect of the posterior septum; Defect of the anterior septum; Defects in other uncommon situations.*

COMPLETE ABSENCE OR VERY IMPERFECT INDICATION OF THE AURICULAR AND VENTRICULAR SEPTA.—The heart consists of two cavities, an auricle and a ventricle, with a single vessel which supplies both the systemic and pulmonic circulations.

Many cases of this kind have been collected by Dr. Peacock, the specimens showing examples of hearts in very different stages of development. One of the earliest records of this malformation was brought before the Royal Society by Mr. Wilson in 1798. The heart was contained in a sac which rested upon the surface of the liver; the lower part of the pericardium was absent. There was a single auricle and ventricle, and one vessel which divided into two branches; the smaller of these went to the lungs, and the other passed upwards behind the thymus gland and gave off the usual aortic vessels. There was no ductus arteriosus, and the two pulmonary veins entered the descending vena cava.

Other cases are also described, by Dr. Farre and by Mr. Forster, in which the heart retained its most rudimentary form.

Examples in which there was some division between the auricular or ventricular cavities have been not infrequently recorded; in some the auricles are more or less divided, but there is only one orifice of communication between these and the ventricle; in others the arterial trunk is divided into an aorta and pulmonary artery.

DEFECTS IN THE AURICULAR SEPTUM.—**Defects of the primary septum.**—*Complete, or almost complete, defect of the interauricular septum.*—The auricle remains single and undivided, or there may be a slight indication of a septum in the form of a sickle-shaped membrane at the upper and hinder part. This condition is usually associated with other considerable abnormality.

Partial defect with open or closed foramen ovale.—There may be a large defect in the lower part of the septum limited below by the upper and hinder part of the ventricular septum, while the foramen ovale is closed and may be seen above the aperture of defect; in other cases the foramen ovale remains open. The pulmonary artery in many of these cases is wider than the normal, and the aorta may be contracted. The result of this form of defect is to leave open a free communication between the auricles and the upper part of both ventricles over the ventricular septum. A specimen of this form of defect is described by Dr. Norman Moore. The auricles were enormously dilated; the apex was bifid like the heart of a dugong. The foramen ovale was completely closed, the septum auriculorum did not meet the septum ventriculorum, and there was a large opening below it, but above the flaps of the mitral and tricuspid valves; one part of each of these was attached to the septum ventriculorum just below this opening; thus the auricles were in communication with one another, and each auricle with both ventricles.

Another specimen is described by Peacock in which, in addition to the septal defect in the auricles, the trunk of the pulmonary artery was dilated, and the aortic orifice was very small.

Defect in the secondary septum.—The septum may be deficient either with or without remains of the primary membranous septum.

The remains of the primary membranous septum may be in the form either of a lattice-like membrane, or a pouch-like sacculation which protrudes into the auricular cavity.

In some instances a defect is found above the foramen ovale, this latter being closed or open. A few cases of this kind are described by Rokitansky.

A case is recorded by Professor Greenfield in which there was a deficiency of a great part of the upper and anterior portions, and in addition a perfectly formed but widely patent foramen ovale. The auricles were enormously enlarged and the appendices elongated, the left coming right round to the front of the heart. When opened the auricles were found separate at the lower part only, and communicated partly

A specimen of this malformation was removed by myself from a girl aged sixteen, who died of pulmonary phthisis. The heart consisted of two auricles and a single ventricle, and the pulmonary artery and aorta were transposed. The septum between the two auricles was complete, but the right was nearly twice as capacious as the left. The coronary sinus opened into the right auricle, and the right auriculo-ventricular valve was tricuspid in shape; the left auriculo-ventricular valve was somewhat irregular, the aortic cusp being puckered and contracted. The single ventricle was capacious, and presented only the merest rudiment of division in the form of a muscular projection at the posterior and inferior part. The aorta was of large size, but arose from what would be the normal position of the pulmonary artery; the aortic valves were normal, also the openings of the coronary arteries. The pulmonary artery arose behind and slightly to the left of the aorta, the opening into the ventricle being situated between one of the segments of the tricuspid and mitral valves. The pulmonary valves were normal, but the orifice appeared somewhat smaller than usual. The ductus arteriosus was closed.

Partial defect of the ventricular septum.—Following the description given by Rokitansky, the ventricular septum may be divided into a posterior muscular septum, a membranous portion, and an anterior muscular septum, the latter being again divisible into a front and hind portion. (See Section II.)

Defects may be seen at one or other of these sites at the base, where during fœtal life the division of the cavities is last effected.

Defect in the posterior septum throws the two ventricles into free communication. A case of this kind is described by Rokitansky; the aperture was of considerable size, and, as seen from the right ventricle anteriorly, opened into the left ventricle, over the free edge of the rudiment of the ventricular septum; the septum of the auricles was incomplete. The free upper edge of the rudimentary ventricular septum was sickle-shaped, and the front portion terminated above in a band which was inserted between the two arterial trunks. The pars membranacea was also defective.

Other cases of similar defect are recorded, associated with abnormal size of the right ventricle, persistent ductus arteriosus, or transposition of the right and left hearts.

Defect in the pars membranacea, or the "undefended space," is ascribed by Peacock as the cause of almost all the apertures found in the upper part of the ventricular septum, and in this he has been followed by many English writers. It is probable that Peacock included in the defects of the pars membranacea apertures which extended both in front of it and behind it. He remarks that if the interventricular septum be partially defective, the imperfection most generally occurs at the base. In this situation there exists normally, in the fully developed organ, a triangular space in which the ventricles are separated only by the endocardium and fibrous tissue on the left side, and by the lining membrane and a thin layer of muscular substance on the right. Laterally it is bounded by the attachments of the right and posterior aortic cusps, and its base is formed by the muscular substance of the septum. The dimensions of the space vary with the size of the heart, but ordinarily in the adult the sides may be estimated at about seven Paris lines, and the base is somewhat wider. When the lower part of the space is perforated, the left ventricle and origin of the aorta communicate with the sinus of the right ventricle, but if the defect be situated high up, towards the angle of attachment of the valves, the communication may be between the left ventricle and the right auricle.

The anterior part of this opening would therefore correspond with an aperture due to defect in the hinder part of the anterior septum as described by Rokitansky.

An aperture confined to the "undefended space" would be of very small dimensions, but it may be defective in conjunction with defects of either the posterior septum or of the hinder portion of the anterior septum.

Complete defect of the anterior septum.—Several instances of this condition are described and figured by Rokitansky. In these the whole of the anterior portion is deficient, throwing both the ventricles and the origin of the arterial trunks into communication.

The majority of these cases showed in addition either transposition or some anomaly in the position of the large arterial trunks. In others there was stenosis or atresia of the pulmonary artery. The foramen ovale was usually open or only partially closed.

Defect of the hinder portion of the anterior septum.—This is a very common form of deformity, and like the rest is usually accompanied by malformation of other parts, with abnormality of the origin of the arterial trunks, or with stenosis or atresia of the pulmonary artery. An aperture in the hinder part of the anterior septum places the two ventricles in communication, the left ventricle and origin of the aorta with the sinus of the right ventricle.

A large number of cases and specimens are described by Rokitansky and others. The aperture has for its posterior limit the pars membranacea.

Defect of the foremost part of the anterior septum.—By this malformation the origins of the arterial trunks are placed in communication; the condition is no doubt rare. Peacock remarks that occasionally, though, so far as his observation serves him, very rarely, the division between the left ventricle and the infundibular portion of the right is perforated, so as to form a communication between the left ventricle and the origin of the pulmonary artery; he also mentions that there are two specimens illustrating this condition in the Museum of St. Thomas's Hospital.

Dr. Sidney Coupland describes an excellent example of this rare form of defect. The heart was hypertrophied, both ventricles enlarged and the walls thickened. On laying open the conus arteriosus the upper part of the ventricular septum was seen to be perforated by a crescentic aperture, which was of sufficient size to admit a No. 12 catheter, and was seated on the posterior wall of the conus, immediately below and to the right of the posterior segment of the pulmonary valves. Viewed from the left ventricle the aperture had the following relations :—Its shape was more oblong than it appeared on the right side, and it occupied the fleshy part of the septum about a quarter of an inch from its union with the anterior wall of the ventricle. The upper margin was formed by the bulging segment of the anterior, sometimes called right aortic cusp, from above which issued the right coronary artery.

The orifice was thus placed between the anterior or right and the left posterior, or left valve cusp, but in closer contiguity to the former than to the latter. There was no further malformation of the heart.

Two cases are described by Rokitansky. In one there was a rounded orifice in the foremost part of the anterior septum on the left side; it was situated beneath the right aortic valve 10 mm. in front of the membranous septum: seen from the right side, it appeared in the conus 13 mm. in front of the membranous portion just below the right pulmonary valve. The apex of the heart was bifid, the aorta displaced to the right, and the position of the pulmonary valves was altered. The aorta and pulmonary artery were of normal calibre.

Defects in uncommon situations.—It is rare to find apertures of communications between the ventricles elsewhere than at or near the base of the septum.

Rokitansky records a case in which, with other malformation, there was a perforation near the middle of the septum. Sir Dyce Duckworth describes a specimen in which there was an aperture in the septum of the ventricles about the junction of the middle and lower thirds; the opening was large enough to admit a crow quill, and was situated somewhat posteriorly; the foramen ovale was pervious. Apertures in these unusual situations do not seem to admit of any general explanation.

STENOSIS OR ATRESIA OF THE PULMONARY ARTERY.—**Stenosis of the pulmonary artery.**—A pronounced example of this the commonest form of cardiac malformation well merits description. The following specimen was removed by myself from the body of a child, aged five and a half years, who died of cerebral abscess.

The heart weighed five and a half ounces; there was marked hypertrophy of both ventricles, more especially of the right. On opening the right auricle it was found to communicate very freely with the left through the foramen ovale. The pulmonary artery was much diminished in size, and there was extreme stenosis of its orifice which admitted the passage of a cylinder only about 8 Paris lines in circumference. The pulmonary valves were only two in number: close to the ostium there were signs of slight recent endocarditis. The septum of the ventricles was incomplete at the upper part just in front of the pars membranacea. The aorta, which was much dilated, was situated more to the right than normal, it communicated freely with both ventricles, rather more with the right than with the left. The ductus arteriosus was not found.

Numerous instances are recorded of this condition; namely, stenosis of the pulmonary artery, imperfection of the ventricular septum, a dilated aorta communicating freely with both ventricles. Minor variations depend on the degree of stenosis, the extent of the septal defect and the degree of displacement and dilatation of the aorta: the foramen ovale and the ductus arteriosus may be either patent or closed.

In a large number of these cases there is some deviation of the septum of the ventricles, so that the origins of the aorta and pulmonary artery are misplaced; this deviation of the septum is most frequently to the left, so that the right ventricle is of large size and the aorta arises wholly or to a great extent from that cavity.

A case of this kind is described by Dr. Parker. The heart had been removed from a boy, aged thirteen, who died of pneumonia. The valves of the pulmonary artery were adherent; the ascending aorta was much dilated, and arose from the large hypertrophied right ventricle. The left ventricle formed only a small supplementary sac with a communication into the right ventricle. In some instances the septum of the ventricles is found to be entire while the auricular septum is defective.

Atresia or obliteration of the pulmonary artery is a far rarer condition than the preceding. Several cases, collected from various sources, are quoted by Peacock; and he records two cases which came under his own notice. An important distinction in these two cases is that in the first the ventricular septum was incomplete, while in the second it was fully formed. In the first there was obliteration of the orifice and trunk of the pulmonary artery; the aorta arising chiefly from the right ventricle and giving off the pulmonary branches through the ductus arteriosus. The right auricle was large and its valves thick, and the foramen ovale was not completely closed by the valve, but would allow the blood to flow from the distended right auricle into the left. The cavity of the right ventricle was of very large size, and consisted almost entirely of the sinus; the infundibular portion was reduced to a mere chink, and was entirely closed at the usual point of origin of the pulmonary artery, the trunk of which formed an impervious cord as far as its union with the ductus arteriosus; the septum of the ventricles was imperfect at the base; the wall of the right ventricle was extremely thick, and the left auricle and ventricle were very small in relation to the right. The aorta arose chiefly from the right ventricle, and was of large capacity so far as the point at which it gave off the ductus arteriosus, through which the supply of blood was transmitted to the lungs.

The second specimen was removed from a child which died nine days after birth. The heart was of unusual form, being broader from side to side than from above downwards. The left ventricle constituted the largest part of the organ. The two auricles communicated freely through the patent foramen ovale. The cavity of the right ventricle was of very small size. The outlet from the ventricle by the pulmonary artery was entirely closed by the union of the valves at the origin of this vessel.

The pulmonary vessel was pervious down to the valves. The ductus arteriosus was of the usual size, and passed into the aorta, forming a communication between the branches of the pulmonary artery and that vessel. The septum of the ventricles was entire. The cavity of the left ventricle was of large size, and was separated from the left auricle by the usual valves. The ascending aorta was large and the ordinary branches arose at the arch. After the entrance of the ductus arteriosus the aorta diminished considerably in capacity. The course of the blood in this case must have been from the right auricle into the left auricle, thence into the left ventricle and aorta, and from that vessel to the lungs by the ductus arteriosus. The right ventricle, being thrown out of use, had atrophied; while the left, having to maintain both the systemic and pulmonary circulations, was unusually capacious and hypertrophied.

A remarkable instance of this condition is recorded by Dr. Hare. It was removed from a child aged nine months, who died cyanotic. The right auricle was enlarged, and had only a very small communication with the left through an opening in the foramen ovale, one-sixteenth of an inch in breadth and one-tenth of an inch in length. On cutting into the right ventricle it was found that the columnæ carneæ were fused almost into one and the cavity would only hold a moderate-sized pea. The ventricular septum was perfect. The orifice of the pulmonary artery was closed, but its trunk was in communication with the ductus arteriosus and divided into the usual branches. The left ventricle was hypertrophied and gave origin to the aorta. The unusually small opening between the right and left auricles, the only communication between the two sides of the heart, was remarkable in this case.

In all cases of atresia of the pulmonary artery the possibility of the circulation being carried on, and life maintained, depends upon the open condition of either the interventricular septum or the foramen ovale; or on the patency of the ductus arteriosus.

There are some important differences in the site of the constriction, partial or complete, of the pulmonary artery, and the nature of the constriction varies also.

The following forms may be recognised:—

Stenosis or atresia of the trunk of the artery.

Stenosis at the conus arteriosus.

Stenosis of the valves with or without narrowing of the trunk of the vessel, and with dilatation of the pulmonary artery.

Stenosis of the trunk.—The trunk and canal of the artery may be contracted or obliterated for a greater or less extent in its course, or even converted into a fibrous cord. The cause of this contraction is no doubt due, in the majority of instances, to irregularity in the development or division of the common arterial trunk, and is usually associated with other developmental defects. Atresia occurs whenever the deviation of the septum of the bulb is so considerable that the septum, the convexity of which is directed towards the pulmonary artery, becomes actually applied to the wall of the vessel and fuses with it as far down as its mouth. The cause of the incomplete division is probably due to imperfect development of the fifth branchial arch.

Stenosis at the conus arteriosus.—The conus or infundibular portion of the ventricle is usually ill developed, and there is a constriction between it and the sinus of the ventricle. The degree of stenosis may be extreme, the orifice being only of sufficient size to admit a small probe. The condition is usually associated with much thickening of the endocardium and surrounding muscular tissue, with increase of the fibrous tissue; these results being in many cases due to the impediment of the passage of the blood of some duration.

There is often some evidence of recent endocarditis about the stricture in the form of roughening or small vegetations.

Stenosis at the valves.—When the constriction is at the valves, their free edges or adjacent parts are adherent, forming a curtain, and leaving an aperture of varying size and shape for the passage of the blood.

The valves themselves are usually irregular in number, size, or form.

The pulmonary artery is usually found to be more or less diminished in calibre throughout; but this is not invariably the case, for in some specimens dilatation occurs in the calibre of the vessel on the distal side of the obstruction. A specimen of this latter condition of the pulmonary artery is described by Peacock. The heart weighed about nine ounces: the anterior surface was almost entirely composed of the right ventricle, which was greatly dilated and hypertrophied. The pulmonary orifice was very much constricted from disease of the valves; the three curtains were blended together so as to form a kind of diaphragm which extended across the orifice, and protruded forwards in the course of the vessel, and was perforated in the centre by a small rounded aperture. The trunk of the pulmonary artery was of somewhat large size, and its coats were thick. The fœtal passages were completely impervious. The case was an uncommon one, for with extensive disease of the valves of the pulmonary artery the heart was otherwise well formed. It must be concluded that the degree of obstruction at the pulmonic orifice must at the time of birth have been only slight. With regard to the dilatation of the trunk of the pulmonary artery combined with the stenosis, Peacock remarks that this is generally the case where the septum of the ventricles is entire, but where the septum is deficient and the stenosis at or near the orifice, the trunk of the artery is usually small and its walls thin.

In cases of obliteration of the pulmonary artery the blood is usually transmitted to the lungs from the aorta through the ductus arteriosus; more rarely from the left subclavian artery or from other branches from the descending aorta.

ATRESIA OR STENOSIS OF THE AORTA.—This may occur either alone or associated with other deformities. A case is recorded by Mr. Shattock of atresia of the aortic aperture in an infant from adhesion of the valves. The ascending aorta was much diminished in calibre, and arose from the left ventricle, the cavity of which was almost obliterated and could only hold a pea. The right side of the heart was large and the ductus arteriosus was patent.

Dr. Peacock mentions a case of obliteration of the aortic orifice, reported by Romberg, in a child who lived four days and was cyanosed. The right ventricle was dilated and hypertrophied, and the pulmonary artery was large. The left auricle and ventricle were very small, and there was not a trace of the aortic orifice. The foramen ovale was largely open, and the supply of blood to the aorta was conveyed from the pulmonary artery by the ductus arteriosus.

Similar specimens have been exhibited by Mr. Canton and by Dr. Hare. In these cases of atresia with complete ventricular septum, the left ventricle becomes abortive, and is almost entirely thrown out of the circulation, and they may be well compared with similar cases of atresia of the pulmonary artery in which the right ventricle becomes abortive.

Rauchfuss has collected twenty-four cases of stenosis and atresia of the aorta, with perfect ventricular septum; it appears that atresia of this orifice is less rare than a similar condition of the pulmonary artery.

Stenosis occasionally affects the left conus arteriosus, but not so frequently as the right.

Stenosis of the arch of the aorta at the ductus arteriosus.—A narrowing of a part of the aorta in this region is sometimes found: a specimen in the Cambridge Museum shows the arch of the aorta to be much contracted from the orifice to the ductus arteriosus, which latter vessel is patent; the aorta then widens to its natural size. It is noteworthy that in the normal fœtus the aorta is considerably reduced in size after giving off the large vessels, that it often presents a marked constriction at the part corresponding to the attachment of the remains of the ductus arteriosus, and that this constriction or isthmus is succeeded by a fusiform dilatation, *the aortic spindle of His.* When the aorta distal to the left subclavian artery is contracted or impervious the descending aorta is usually wholly or chiefly supplied through the pulmonary artery.

A curious case of aortic stenosis, with other defects, is recorded by Dr. Greenfield. The heart was greatly enlarged, especially the right ventricle; the two auricles communicated freely; the septum of the ventricles was entire. The left ventricle was somewhat hypertrophied and dilated: the aortic valve consisted of two cusps, anterior and posterior, the anterior being formed by the fusion of two. The aortic orifice was greatly narrowed, and the aorta commencing a little beyond the valve showed marked dilatation. The ductus arteriosus was closed, and beyond its point of junction the aorta became narrowed, and then again returned to its normal size; the pulmonary artery was dilated.

Hypoplasia of the aorta with smallness of the heart was described by Virchow in 1856 in connection with chlorosis: more recently Beneke made elaborate measurements of the vessels at different periods of life, pulmonary artery was represented by a fibrous cord, closely adherent to the aorta, which could be traced down to the flattened elevation of the ventricle before mentioned. The two pulmonary arteries received their blood-supply by a patent ductus arteriosus, and the lungs were further supplied with blood by the greatly enlarged bronchial arteries. The position of the left auricle was especially noteworthy in this case, having been, as it were, dislocated behind the aorta and rudimentary pulmonary artery. Professor Griffith remarks that it is difficult to avoid the conviction that it may, by pressure, have prevented the development of the proximal part of the fifth right branchial arch, and thus led to an almost total absence of the main stem of the pulmonary artery.

In another specimen, described by the same author, there was lateral and antero-posterior transposition of the aorta and pulmonary artery. The heart was somewhat enlarged, the ventricular part being especially bulky. The two auricles were normal in most respects, but the foramen ovale was widely patent—the deficiency being above and in front of the valve, which was also defective at its upper and anterior part. On opening the ventricular cavities they were found to communicate freely with one another by a large aperture at the upper part of the septum, limited below by a smooth crescentic-rounded margin. The posterior boundary of the opening was continued up as a thin fibrous membrane, and blended with the upper part of the septal flap of the right auriculo-ventricular valve, which it separated from the orifice of one of the vessels arising from the ventricular cavity. There was thus an absence of the anterior part of the septum which is developed from the aortic bulb septum, while the posterior part, derived, according to His, from a septum medium, was normally developed; the ventricles were not transposed. From the upper and anterior part and from the right ventricle arose a vessel which arched backwards over the root of the right lung, and was continued down the back of the chest. It gave off the coronary arteries and vessels to the head and upper extremities; from behind this aorta arose another vessel from the ventricular cavity, which gave off the branches to the lungs and then joined the arch of the other large vessel; a patent ductus arteriosus also connected the two vessels. The second vessel, therefore, appeared to have the mixed characters of the aorta and pulmonary artery. The valves of this vessel formed a bicuspidate cone projecting into the lumen.

An unusual form of transposition of the primary vessels was found in a case by Dr. Hess. It was removed from a child eight hours old, who died with coma and convulsions. The heart was quadrangular in shape, the auricles were completely separated, and both auricles opened into the left ventricle. The left ventricle was very large, and at the upper and posterior part gave origin to the pulmonary artery. The right ventricle was a small rudimentary cavity from which the aorta arose, and which communicated with the left ventricle by a crescentic opening ten lines in circumference; apparently the sinus and infundibular portion of the right ventricle were divided by a septum, from the latter the aorta was given off, while the sinus was united with the left ventricle from which the pulmonary artery arose.

Other forms of malposition are recorded, though far less frequently, in which the two vessels arise from the left ventricle, while the right ventricle is merely a rudimentary cavity, and has communication with the left through an aperture in the septum.

PREMATURE CLOSURE, OR PATENCY OF THE FŒTAL PASSAGES.—**Premature closure of the foramen ovale.**—This condition is extremely rare; there are only three cases recorded by Peacock; in one the child lived thirty hours, and was cyanosed, the right ventricle and pulmonary artery were extraordinarily developed, and there was no trace of the foramen ovale. In the other two cases, which were similar as to the obliteration of the foramen ovale, the right cavities were greatly enlarged, but the left were on the other hand very small.

Patent foramen ovale. See Defects in the auricular septum, p. 699.

Premature closure of the ductus arteriosus.—The duct may become abortive at different periods of fœtal life, judging from the fact that in some malformed hearts no remains of it can be found. In such cases the pulmonary artery is usually narrow and ill-developed, owing to the small quantity of blood which circulates to the lungs in fœtal life. The obliteration of the duct is probably due to imperfect development of that portion of the branchial arch, and may be one of the causes of pulmonary stenosis. Other deformities usually coexist or supervene as the result of the premature closure of the duct.

Persistency of the ductus arteriosus is the result of failure of the normal involution which usually takes place before the fourteenth day. The vessel may be widely patent or funnel-shaped, and in the majority of cases the orifice of the pulmonary artery is stenosed or closed. The right ventricle is hypertrophied, and the trunk of the pulmonary artery may be dilated. In a few instances the duct has remained patent without other anomalies.

IRREGULARITIES IN THE NUMBER AND FORM OF THE VALVES.—Slight defects in the semilunar valves are of comparative frequency and do not cause any symptoms; they may be due to malformation or to fœtal endocarditis. The number may be reduced or increased.

Bicuspid semilunar valves.—This, the commonest form of anomaly, in which there are only two segments, affects both the pulmonary artery and the aorta. One segment is sometimes normal in size, the other, frequently the larger, appears to be the result of the union of two segments, showing often an indication of the division between them; or the two may be of nearly equal size.

There may be only one curtain, with an indication of its division into three segments; it becomes stretched or protrudes in a funnel shape in the course of the vessel. Rarely there are two large segments with a small rudimentary one interposed.

The bicuspid form of valve has a great tendency to undergo sclerotic change, and to result in regurgitation. In the aorta it has been noted that the segments united are not infrequently those opposite the coronary orifices. In many the result is due to malformation, but endocarditis may account for some of those formed in later life, the partition between the two segments having been destroyed. When the pulmonary valve is anomalous there is usually found some other malformation, such as septal defect.

Redundancy in the number of segments more frequently affects the pulmonary artery than the aorta. The chief forms are (i.) three of nearly equal size, with a smaller one interposed between two others; (ii.) four segments of nearly equal size; and (iii.) three or four segments of nearly equal size with one or two smaller curtains interposed, and imperfectly separated from those adjoining.

The valvular anomalies due to mal-development take place at the time that the aortic bulb is transformed into aorta and pulmonary artery. Where the number of segments is deficient there is probably suppression of one of the endothelial cushions. On the other hand, when there is redundancy of the segments one rudiment gives rise to two or more segments. This most commonly happens in the case of the external rudiment, the last to appear.

The auriculo-ventricular valves.—The segments of the tricuspid or bicuspid valve are sometimes found united together in the form of a membranous curtain with a central triangular or circular aperture. In some the stenosis is no doubt due to fœtal endocarditis or malformation; in others it is very difficult to determine whether subsequent endocarditis of sclerotic origin may not account for the greater part, if not all, of the resulting lesion. The two apertures may be affected in the same heart, and with a history of long-standing cyanosis in a young person, and in the absence of rheumatic attacks, it is almost certainly of congenital origin. The united and malformed cusps are very liable to become the seat of disease, and the stenosis is increased by chronic thickening of the united valve segments, but vegetations are seldom found.

ANOMALOUS SEPTA.—The majority of cases in which supernumerary cavities in the heart are described are really due to the existence of an anomalous septum. This is most commonly found in the interior of the right ventricle, and at a site where there is normally a strong muscular band indicating the division between the sinus and the infundibular portion of the right ventricle. In well-marked cases there is a distinct resemblance to the right systemic and pulmonic ventricles of the turtle.

There is usually an aperture of communication between the middle and right ventricles, but the right ventricle has no direct connection with the auricle. Two cases are recorded, by Dr. Stephen Mackenzie, in which there were, in addition to many other abnormalities, apparently three ventricles; he remarks that the infundibulum of the right ventricle was shut off from the sinus by means of an imperfect, partly muscular septum, an exaggeration of the division of the muscular columns to which the folds of the tricuspid valve are attached.

Septa or fibrous bands are more rarely found in the auricles. Dr. Rolleston and Dr. Wardrop Griffith record such anomalies occurring in the left auricle. Dr. Fowler describes a similar instance, in which there was a band attached to the septal wall and continuous with the membrane forming the fossa ovalis. He regarded this band as an overgrowth of the valve closing the foramen ovale which had become directed by the blood-stream towards the outer wall of the auricle, and had become adherent there.

The so-called moderator bands, which are occasionally found in the interior of the ventricles, consist of muscular fibres surrounded by endocardium. They not infrequently arise from the septum, and are attached to the wall of the ventricle. In a case recorded by Sir William Turner the inner surface of the ventricles was almost uniformly smooth.

GENERAL ANOMALIES.—Some of these occur in monsters which are still-born.

External misplacements.—*Ectopia cordis.*—*Clefts of the thoracic wall* and fissure of the sternum may be present, so that the heart is covered only by membrane and integument, and protrudes; in other cases there is no apparent defect of the thoracic wall. There is commonly some other malformation present, such as protrusion of the abdominal viscera.

Three varieties are usually described: ectopia cervicalis, pectoralis, and abdominalis. In the first the heart is placed in the neck, in close connection with the ramus of the jaw. In the second form there may or may not be a fissure of the parietes of the chest. In the abdominal form the organ lies below the diaphragm, and is sometimes protruded so as to form a tumour externally. In one well-noted case the heart was found to occupy the position of the right kidney, and the vessels arising from it passed through the opening in the diaphragm into the thorax.

Internal misplacements.—*Dextro-cardia.*—Transposition of the heart is generally associated with transposition of the viscera. A few cases have been observed in which the transposition affected the heart only.

Two hypotheses have been proposed for the explanation of this anomaly. Dr. Frazer suggests that the transposition may be due to the subject having been one of twins which were developed from a single ovum, and in which dichotomy was complete. Von Baer has found that in a few instances the embryo lies with its left side directed towards the yolk, whereas the right side is normally in this position.

Meso-cardia.—The organ occupies a central position in the thorax similar to that which obtained at the earlier periods of fœtal life. It usually presents anomalies in structure as well.

Bifid apex.—Occasionally there is an indication of a fissure at the apex of the heart, following the course of the interventricular septum, and more or less dividing the apex into two, giving a resemblance to the heart of the dugong.

Deficiency of the pericardium.—Complete absence of the pericardium is very rare except in association with ectocardia, or other serious anomaly. Partial defect is sometimes observed, and the only remnant of the pericardium may be found in the form of a sickle-shaped fold attached to the diaphragm which forms an incomplete sac for the heart. A specimen was described by Dr. Bristowe, in which there was a rudiment of the pericardium at the upper part and right side of the heart. In another case, recorded by Dr. Boxall, the pericardial sac was incomplete, and death was caused by dislocation of the heart during a severe attack of vomiting.

SECTION II

CAUSATION

SYNOPSIS.—*Fœtal endocarditis—Mal-development—Embryonic heart—Mode of formation of septal defects—Stenosis and transposition.*

The cause of the various forms of cardiac abnormality is an interference with the normal processes of development at some particular stage of embryonic life. Thus, an arrest of development may occur in which the heart retains in great measure the rudimentary form of the stage at which its growth is arrested; or there may be some perversion or irregularity in development at some part by which distortion is produced, and which gives rise to secondary changes dependent on the primary defect.

In some cases in which the malformation has occurred at a very early date, as for instance where the heart consists of only two cavities, it may be impossible to detect the primary deviation from the normal. In many, however, where the heart has been more fully developed, it is often possible to detect the primary defect, or, at any rate, to trace the sequence of events by which the secondary changes have been induced. Fœtal endocarditis and mal-development or perversion of the processes of development are responsible for most abnormalities.

An attack of rheumatic fever in the mother during pregnancy, or a tendency to rheumatism in the parents, may be a cause of fœtal endocarditis; but in most instances no such history can be obtained. The arrest of development has been attributed by some to maternal impressions during pregnancy, but in many cases the date of the impression does not coincide with the period of fœtal life at which the arrest must have taken place.

Fœtal endocarditis has by some writers been credited with a large share in the production of different forms of cardiac malformation, and probably to a far greater extent than is justified by the evidence.

The chief form of inflammation of the fœtal endocardium is of the sclerotic kind; the warty form is of far less frequency, although it is seen occasionally affecting the edges of the adherent and stenosed pulmonary or aortic valves. Minute projections may be found on the auriculo-ventricular valves of newly-born children; these have been mistaken for vegetations. They consist of nodules of translucent or firm connective tissue which usually disappear in the course of time. In others the edges of the valves, more often the mitral, are the seat of hæmatomata, caused by small spherical blood extravasations projecting from the free edge of the valve, and probably due to the rupture of intravalvular blood-vessels. They seem to arise either before or shortly after birth, and very soon shrink away; occasionally they are found in connection with a stenosed valve. In the sclerotic form the cusps are thickened and contracted, and the edges often united to those adjoining; the chordæ tendineæ become thickened, and the valvular orifice much diminished in size. It is often impossible to tell whether the endocarditis is of fœtal origin, or has at a later period become engrafted upon an already deformed valve. According to Rauchfuss, fœtal endocarditis is only more common on the right side of the heart when in association with malformation, otherwise the left heart is as frequently affected.

Perversion of development.—Interruption to the normal course of development is the cause of the greater number of cardiac malformations. This is in great measure indicated by the nature of the defect, the early period of fœtal life at which the first deviation must have occurred, and by other circumstances which tend to show that if any endocarditis is present it has been engrafted upon an already deformed valve or orifice. This view is strengthened by the observation that in a considerable number of instances developmental errors are present in other parts of the body. Dr. Archibald Garrod has collected a series of eighteen such cases, the associated abnormalities being of various kinds. In five of the eighteen cases fœtal endocarditis was clearly present, but in three of these there were other abnormalities which were obviously not secondary to the inflammation; in two the associated defects were of a minor kind, and fœtal endocarditis sufficed to explain all the appearances. But even if malformation be regarded as the primary cause, we still remain in ignorance of the nature of the force which disturbs the natural process of evolution.

Before attempting to discuss the mode of formation of the various specimens of malformation described in Section I., it will be necessary to refer to the development of the embryonic heart. A full account would be out of the scope of this article, and attention will only be drawn to those events which help to elucidate the pathology of the malformed specimens.

Development of heart.—The heart is originally developed out of two lateral tubes of mesoblast, symmetrical and distinct, which coalesce, soon after the thirteenth day, to form a single longitudinal tube, which is slightly twisted upon itself. This single tube has double walls, the inner endothelial, the outer mesoblastic or muscular; it is continuous in front with the two primitive aortæ, and posteriorly with the veins. During the third week slight constrictions become evident which mark off the several divisions from one another. The anterior of these is the aortic bulb, the middle thicker part is the ventricular portion, and the posterior forms the auricular segment.

This tube then becomes bent upon itself in such a way that the venous or auricular portion comes to lie partly dorsal to, and partly behind the ventricular portion, the latter being continued forward as the bulbus arteriosus. Between the primary undivided auricle and ventricle a constriction occurs which elongates into a short flattened canal, the auricular canal, which is bounded by two lips, an upper and a lower. These lips become thickened by the formation of endocardial cushions which grow across the canal in such a way as to divide it into two passages, the right and left auriculo-ventricular orifices.

The internal division of the heart into right and left sides is effected by three septa or partitions, which appear within the cavity of the heart, and which arise perfectly independently of one another; namely, the interauricular septum, the interventricular septum, and the septum of the truncus arteriosus.

The interauricular septum.—The division of the auricle precedes that of the ventricles and of the bulb. The history of the process as given by His, Lindes, and Born differs in some important respects. According to Lindes and Born, when the auricles develop they expand upwards, and a partition remains between them at the upper part, the septum primum, or septum superius. This septum increases with the continued growth of the auricles, and becomes thickened along its lower edge, and finally separates the two auricles, except under its lower edge, where the two cavities still communicate. This communication is not, as has been previously maintained, the foramen ovale, inasmuch as the septum continues to grow downwards to the auricular canal, and, by uniting with the partition in the canal, closes permanently the primary communication. According to Lindes, before the primary septum has quite reached the roof of the ventricles, certain small apertures may be noticed in it. These gradually increase in number, converting the septum into a lattice-like membrane through which the blood streams from right to left, causing the septum to bulge to the left.

The parietal portion only of the septum remains imperforate, forming a muscular frame which is especially well developed anteriorly. Finally, there is one large aperture left in the septum at its apex and anterior part, the true foramen ovale. A new septum also appears above the foramen ovale and to the right of the insertion of the primary septum, and its edge forms part of the boundary of the foramen ovale. In a human embryo 25 mm. long, the auricular septum contains numerous perforations, and in a fœtus of three or four months the septum appears as a cribriform membrane supported on a muscular frame. During the fourth month the foramen ovale becomes partially closed by a fold which acts as a valve and allows the blood to pass from the right to the left auricle, but prevents its passage in the reverse direction. The final closure of the foramen ovale does not take place until some time after birth, and is one of the last events; it is at first effected merely by the close apposition of the valve which projects into the left auricle to the margin of the aperture by the pressure of the increased quantity of blood returning by the pulmonary veins; at a later stage the edge of the valve gradually coalesces with the margin of the opening, but the union often remains incomplete for some months.

The ventricular septum and division of the truncus arteriosus.—The ventricular cavity becomes partially divided towards the close of the fourth week by a fold, the septum inferius, which rises from its dorsal and posterior wall, and the position of which is indicated externally by a slight groove on the surface of the heart.

The formation of the aortic septum is effected by two longitudinal ridge-like thickenings of the endothelial lining which arise from opposite sides at the junction of the fifth branchial arch; these encroach on the lumen, reducing it to a slit, dumb-bell in section, and then meet so as to divide the lumen into two completely separate passages.

The septum appears first at the distal end of the truncus, and gradually extends backwards towards the ventricles. The septum first appears towards the end of the fourth week, and is well advanced before the end of the fifth week; it has a slightly spiral course, so that the two tubes into which it divides the truncus arteriosus are respectively dorsal and ventral at the proximal end next to the ventricles, and right and left at the distal end of the truncus. Of the two tubes the one which lies dorsally at its proximal end and on the right side distally is the systemic trunk, the other which is ventral proximally and on the left side distally is the pulmonary trunk; and the same relations are retained throughout life by the ascending aorta and the root of the pulmonary artery.

The *truncus arteriosus* originally arises from the right-hand corner of the ventricular cavity, and the two trunks into which it splits retain for a time the same relations. In other words, at a time when the interventricular septum is already partially formed, both the systemic and pulmonary trunks arise from the right ventricle, and the left ventricle has for a time no outlet except through the right ventricle. The completion of the interventricular septum has to be effected in such a way that while the pulmonary trunk is left in connection with the right ventricle, the systemic trunk shall be cut off from this cavity and placed in communication with the left ventricle. The formation of the interventricular septum is consequently somewhat complicated. The greater part of the septum is formed from the septum inferius, but it is completed above, partly by the endocardial cushion at the lower edge of the interauricular septum, the septum intermedium of His, and partly by the prolongation of the aortic septum, which divides the truncus arteriosus into systemic and pulmonary trunks.

The aortic septum grows back beyond the truncus arteriosus, so as to project a certain distance into the ventricular cavity; it then fuses with the free lower edge of the interauricular septum in such a way as to cut off the systemic trunk from the right ventricle, and to place it in communication with the left ventricle; while finally the septum inferius extends so as to meet and fuse with the interauricular septum, and so completes the separation of the ventricles from each other.

Auricular septal defects.—From the study of the specimens of defect of the auricular septum in connection with its development it will be apparent that apertures may exist either at the foramen ovale or in other parts of the septum. In the latter case, those which exist at the lowest part of the septum are probably due chiefly to failure of union of the primary membranous septum with the upper part of the ventricular septum and with the partition in the auricular canal; thus leaving a free communication between the two auricles and between the latter and the ventricles.

In some cases the septum may be entirely absent, the auricular cavities remaining undivided. When the growth of the secondary septum is defective there is frequently to be seen a lattice-like membrane between the two auricles which imperfectly divides them, and is due to the persistence of a portion of the provisional membranous septum which stretches across the persistent muscular frame. If absent or largely defective it may give rise to an aperture at the upper and front part of the auricular septum; and the completely formed foramen ovale, either closed or patent, may be found below. In other cases the persistent membrane becomes sacculated, and protrudes in a pouch-like form towards the interior of the auricle.

Defects in the ventricular septum.—*Normal arrangement of septa.*—The septum ventriculorum is divided into a posterior muscular septum, a pars membranacea, and an anterior septum; the latter being again separated into a posterior and an anterior portion: the importance of this division is well insisted upon by Rokitansky in his classification of septal defects in the ventricle.

In the higher mammalia the normal arrangement of the septa in the fully developed heart is as follows: the cross-section of the ventricle is that of a crescent, the pulmonary artery being at the anterior extremity of the infundibular portion of the ventricle, while the posterior horn is occupied by the auriculo-ventricular orifice above the sinus of the ventricle. The internal wall is composed of two more or less distinct parts. The anterior is formed of oblique bundles passing from above downwards and slightly from behind forwards. These bundles arise superiorly to the left of the pulmonary artery and pass to the superior half of the anterior margin of the ventricle. They correspond to the false septum of reptiles. Amongst the larger number of mammals the posterior border of this septum forms a very evident projection, or else sends obliquely a fleshy tongue or band to the external wall which accentuates this distinction. This septum is interposed between the pulmonary artery and aorta. The radiating fibres of the rest of the ventricle are placed between the two auriculo-ventricular orifices and the two ventricular cavities. The external wall is covered with fleshy columns arising from the pulmonary orifice, and running obliquely from before backwards and downwards, which establish a limit between the general ventricular cavity or sinus and the infundibulum. At the junction of these two columns with the posterior border of the septum is occasionally seen a white fibrous line or cicatrix. If this spot is perforated by a needle the aorta is penetrated below the right sigmoid cusp.

It is supposed by Sabatier that this cicatrix is the vestige of an orifice from the right ventricle, representing the opening from this ventricle into the left aorta which is present in reptilia. This anterior portion of the ventricular septum is muscular in structure, but immediately posterior to this it will be found thinner and membranous in character; this *pars membranacea septi* or undefended space is more obvious in the heart of an infant than in an adult. Along the upper line of this thinner portion is attached the internal flap of the tricuspid valve. It corresponds to the upper border of the middle portion of the interventricular septum, and behind this again the septum is thicker and muscular in structure.

Reference to the specimens of defect before described shows that apertures in the posterior portion of the septum, in the pars membranacea, or in the posterior part of the anterior septum, will place the two ventricles in communication; while a defect in the front portion of the anterior septum will cause an aperture of communication between the two arterial trunks. The latter defect is much rarer than the other kinds; the aperture is situated below and in front of the right aortic cusp, and perforates the conus arteriosus just below the mouth of the pulmonary artery, and involves the fleshy part of the septum. Rokitansky regards this defect as due to failure in the complete union between the septum of the bulb and the interventricular septum, which takes place at an early period before the completion of the hinder part of the anterior septum.

In many cases where there is a defect at the pars membranacea or at the hinder part of the posterior septum, or an aperture extending into both of these regions, there is a primary defect in the development of the arterial trunks, and the vessels are either misplaced or one of them is stenosed.

Frequently there is evidence of endocarditis surrounding the aperture, and the endocardium is roughened or thickened.

Cases are recorded in which the pars membranacea has been found sacculated and bulging into the cavity of the ventricle, forming the so-called aneurysms of the undefended space, and due in a few instances to congenital weakness at the spot. In some, no doubt, endocarditis has an important share in their formation, and they are due to disease in after-life.

Stenosis or atresia of the pulmonary artery.—This deformity is primarily due either to irregularity in the division of the common arterial trunk or to fœtal endocarditis.

When stenosis occurs at an early period of fœtal life, towards the end of the second month, or early in the third month, when the ventricular septum is well developed but not closed, and the auricular septum is forming, the right ventricle, unable effectually to discharge its contents through the narrow pulmonary artery, becomes over-filled, but is able to relieve itself by outflow over the still unclosed base of the interventricular septum, a measure which is sufficient in itself to prevent the complete closure of the septum. The right auricle in the same way, distended by the backward pressure, finds relief into the left auricle, and thus the normal course of the circulation is materially impeded. When the stenosis is considerable and interferes at a still earlier period with the emptying of the right ventricle, the growing septum becomes pushed over more and more to the left by the distension of the right side, and so prevents the proper connection of the aorta with the left ventricle; and in addition a constant flow of blood is established from the right ventricle into the aorta, so drawing the aortic orifice still farther to the right, and producing a widening of this aperture and also of the ascending trunk of this vessel. To such an extent may this displacement of the aorta be carried that this vessel has origin entirely from the sinus of the right ventricle, the left ventricle being left as a small supplementary sac with a communication into the right ventricle. This is in the main the explanation given by Dr. Hunter, and accepted by the late Dr. Peacock. It is held by some authors that the same series of events might be produced by an irregularity in the division of the bulb, in which the septum descended so as to form a wide aorta at the expense of the pulmonary artery, the aorta being naturally situated farther to the right in the earlier period of fœtal life.

The hypertrophy of the right ventricle in these cases is the obvious result of the large share it has to take in carrying on the systemic circulation through the aorta. When the defect in the interventricular septum is considerable, or the communication of the right ventricle with the aorta very free, the septum of the auricles is more likely to be complete than where the reverse obtains; owing to the less degree of disturbance of the circulation through the auricles.

In atresia or complete obliteration of the canal of the pulmonary artery the obstruction is either due to adhesion of valve segments, an impervious orifice, or obliteration of the trunk of the vessel as far as the ductus arteriosus. The primary defect may occur in early fœtal life before the ventricular system is completed; or later, when the cavities have been separated. In the former case, as in stenosis, the right ventricle retains its communication with the aortic orifice, and is the main agent in carrying on the systemic circulation, while the left ventricle remains small, and atrophies. When the obliteration of the pulmonary artery occurs after the completion of the ventricular system, the right ventricle becomes almost abolished and the right auriculo-ventricular aperture diminished in size. The left ventricle, on the other hand, becomes enlarged, and its walls much hypertrophied, as it has to carry on both the systemic and pulmonary circulations.

In almost all these cases the blood is carried to the lungs by the pervious ductus arteriosus. The foramen ovale is occasionally closed when the ventricular septum is imperfect, but is necessarily open when this septum is complete. Of thirty-four cases collected by Dr. Peacock, in eight only was the ventricular septum completed, and all these latter died a few months after birth.

In all cases of atresia of the pulmonary artery the possibility of the circulation being carried on depends upon the open condition of either the interventricular or the interauricular septum, and the patency of the ductus arteriosus.

Atresia, like stenosis, is probably due to an abnormal division of the bulbus arteriosus. Atresia occurs whenever the deviation of the septum of the bulb from the normal arrangement is so considerable that the septum whose convexity is directed towards the pulmonary artery becomes actually applied to the wall of that vessel and fuses with it as far down as its mouth.

Stenosis or atresia of the aorta.—When the constriction occurs before the completion of the ventricular septum, the narrowing of the aorta must occasion the blood to accumulate in excessive amount in the right ventricle; since both aorta and pulmonary artery communicate originally with this cavity. This repletion of the right ventricle must cause a corresponding repletion of the right auricle, and a distension and enlargement of the passage of communication between the two auricles. If, however, development proceeded as far as closure of the passage through the ventricular septum, and limitation of the aorta on the side of the right ventricle, the condition of repletion would be confined to the cavities of the left heart, and would occasion enlargement in them also.

In atresia of the aorta the left ventricle becomes abortive and is almost entirely thrown out of the circulation; as happens in the case of the right ventricle in atresia of the pulmonary artery.

Transposition or malposition of the aorta and pulmonary artery.—The condition of the cardiac cavities associated with transposition may be perfectly normal, but more commonly shows extensive derangement.

The explanation of these deformities must be found in connection with an abnormal division of the bulbus arteriosus, and the development of the complete septum between the arterial trunks.

The torsion of the axis which takes place during the first seven weeks has a very important bearing; for any departure from the normal, or a failure in bringing the arterial bulb into due relation with the anterior segment of the interventricular septum, is the direct agent in the causation of malposition or transposition of the great arterial trunks. It is probably during the sixth, seventh, or eighth week that these abnormalities first begin. The union of the forked septum which grows down the arterial bulb from above with the upper and fore part of the interventricular septum determines the exact relation of the opening of the two arterial trunks to one another, and the slightest deviation will derange the relation. It should be observed also that the bulbus arteriosus originally communicates with the right ventricle, that it becomes divided into an anterior pulmonary artery and a posterior aorta, at which stage both the large arterial vessels belong to the right ventricle.

The left ventricle would be quite destitute of way of issue, did not the ventricular septum remain permanently open as the aortic orifice. At this period the left ventricle pours its blood into the right, whence mixed blood is driven into both arterial trunks.

SECTION III

SYMPTOMS AND PHYSICAL SIGNS.—A child suffering from congenital malformation of the heart is weakly, difficult to rear, and generally presents at birth, or soon after, signs of derangement of the circulatory system. Lividity, of a bluish-violet tint, affecting especially the face, hands, feet, and the visible mucous membrane, is apparent.

The respiration is often laboured, and paroxysms of difficult breathing may occur from time to time. These are apt to be exaggerated by screaming, struggling, suckling, or exposure to cold air. The extremities are cold and the terminal phalanges of the hands and feet may be clubbed.

From observations made by Farre and Peacock the bodily temperature is not lower than normal, but Henoch and others record considerable lowering of the surface temperature, although normal in the rectum.

Convulsions and cerebral seizures are frequent and often fatal. In a case observed by myself the child was liable to attacks of prolonged unconsciousness. These usually occurred once or twice in the week after a meal, lasted for several hours, and recovery took place without any ill effect; the attack was accompanied by much increase of the cyanosis.

Paroxysms of dyspnœa and palpitation of a dangerous kind are common, in which the breathing becomes rapid, gasping, and noisy, and in which the cyanosis is greatly intensified. Convulsive seizures may be induced, and the attack is often followed by severe exhaustion.

The onset of symptoms is variable; these may be obvious from the first, or there may be no evidence of anything wrong with the child until a year or more after birth, when perhaps the onset of some accidental affection unmasks the latent defect. The earliest and most definite symptom is cyanosis.

Cyanosis.—This is present in about 90 per cent of these cases, hence the origin of the name Morbus Cæruleus.

The pathology of cyanosis in congenital heart disease has from early times occasioned much discussion, and divers explanations have been brought forward to account for it.

The hypotheses ordinarily adduced are those which attribute the condition to intermixture of the arterial and venous blood, or to extensive venous congestion. The former of these is amply negatived by the observation that in many cases of single ventricle no cyanosis has been observed; and that cyanosis may exist without any admixture of the blood-currents.

The admixture hypothesis has been attributed to William Hunter by Peacock and other writers. Reference, however, to Hunter's cases of congenital malformation does not confirm this interpretation. He does not even mention the admixture of the blood as the cause of the cyanosis; but after remarking on the small quantity of blood which reached the lungs in two cases of pulmonary stenosis, he says that, as the carnation tint of complexion depends on the florid colour of the blood, the dark or gray complexion in these cases corresponds particularly with the observations of the latest philosophers that the blood takes its bright hue in the lungs from respiration.

The venous congestion hypothesis, advanced by Morgagni, and ably supported by Stillé in America, has been most widely accepted, but cannot be said to cover the whole field.

It is probable that there are other factors which combine with venous stasis to produce the peculiar discoloration. The possibility of sufficient aeration of the blood through the vessels going to the lungs must be taken into account. Dr. Lees regards this as the essential cause of cyanosis, and estimates that the amount of cyanosis is a direct measure of the extent to which aeration of the blood has been hindered. It must also be noted that it is mainly in cases where obstruction to the circulation has existed before birth, or long before the full development of the circulatory system, that the cyanosis occurs. The condition of the integuments will materially affect the colour; where the patient is emaciated and the skin is thin the peculiar purple or black tint is frequently observed; on the other hand, when the body is well nourished, or the skin œdematous, the colour is of a deep rose tint and less intense.

The blood.—More recently attention has been drawn to the condition of the blood in cyanosis, and Dr. Gibson, in a most interesting paper, discusses the various explanations of congenital cyanosis and draws attention to the remarkable concentration of the blood.

He describes the results of his examination of the blood in a case of this affection: the hæmoglobin was 110 per cent, the red corpuscles were 8,470,000, the white 12,000. He offers in explanation of this concentration the suggestion that in venous stasis the corpuscles are insufficiently oxygenated and their functions imperfectly performed, and that there is less metabolism in the tissues and less waste; consequently, in cyanosis the wear and tear is reduced, and the duration of the individual existence of the red cell is increased. The number must therefore be proportionately augmented, causing a numerical increase and a high percentage of hæmoglobin.

Toeniessen first observed the condition of the blood in a case of congenital stenosis of the pulmonary artery; the red cells were 7,540,000, and in another case 8,820,000. He also noted this marked increase of the red cells in all forms of cyanosis from failing circulation.

Baunholtzer, as the result of examination of the blood in a case of pulmonary stenosis with cyanosis and clubbing, remarks upon the striking concentration of the blood: the hæmoglobin stood at 160 per cent, the number of red cells at 9,447,000 against 5,000,000, the specific gravity 1071·8 instead of 1035-1068.

Dr. Lloyd Jones observes that in the newly-born child the specific gravity of the blood is very high (about 1067); and he has made the same observation in cases in which the foramen ovale had never closed, and in which the fœtal condition of the circulation remains.

The clubbing of the digits consists in a drum-stick enlargement of the terminal phalanges of the fingers and toes, with often a claw-like appearance of the nails. It is usually later in its appearance than the cyanosis, but may be present when cyanosis is absent.

The two symptoms are allied, though possibly not produced under the same conditions. Dr. Lees considers that clubbing is produced by the venous congestion, and remarks that in cases where there was no clubbing there was marked absence of venous congestion.

Cardiac signs.—The detection of cardiac malformation by the physical examination of the heart is usually not difficult; but a diagnosis of the exact form of anomaly must in many cases be impossible.

In some it is possible to arrive at a fairly close decision as to the existing conditions. On *percussion* the heart will usually be found enlarged, with indications of hypertrophy and dilatation of the right ventricle and auricle; the impulse is powerful, displaced outwards and visible over a large area, and there may be some prominence from yielding of the parietes in the precordial region.

On *auscultation* there is commonly to be heard a loud, long, systolic murmur, which can be traced with varying intensity over the whole of the precordial region, over the back of the chest, and is conducted widely in all directions. These may constitute all the cardiac physical signs, and it would be impossible upon these to make an exact diagnosis, inasmuch as they have been found in the most diverse forms of anomaly. There are, however, in one class of cases certain signs which enable us to predict, with a great measure of certainty, the most important anomaly, namely, stenosis of the pulmonary artery. In many of these there is to be felt on light palpation, at about the second left interspace, a fine thrill, systolic in time; it may be appreciable over a considerable part of the precordial area, but is most marked at the upper part; an impulse can often be felt below the xiphoid cartilage; on percussion the dulness extends beyond the right border of the sternum; on auscultation a loud blowing murmur, systolic in time, is also present, and is to be heard louder at the left base than elsewhere. The second sound may be faint or accentuated, or accompanied by a diastolic murmur. With these signs pulmonary stenosis is almost certainly present.

The character of the second sound at the pulmonary cartilage is somewhat variable. In many cases it is feeble and faint; in a few cases which have come under my observation it has been loud and ringing. This ringing sound has attracted the notice of other writers, but its significance has not been ascertained. Garrod reports two cases in which this peculiarity of the second sound was observed, but there was no autopsy. Peacock regards the accentuated sound at the base as produced by the aortic valves, this vessel being often unusually large. On the other hand, it has been suggested that this sign indicates obstruction at the conus arteriosus. The sign is probably not distinctive of the particular seat of obstruction, but it may be due to dilatation of the pulmonary artery immediately distal to the stenosis and a patent ductus arteriosus.

When the pulmonary artery is dilated, with patency of the ductus arteriosus, there may be great increase of cardiac dulness to the left and upwards as high as the second rib; a loud rumbling systolic murmur being audible over the pulmonary cartilage, and an accentuated second sound. Compensation takes place with great readiness, and the right ventricle accommodates itself to the lesion; the possibility of hypertrophy of this chamber at an early age appears to be very great and materially influences the prognosis.

A precise diagnosis of imperfections in the septa is not possible. In these cases a blowing systolic murmur is commonly to be heard over the precordia, which in defects of the auricular septum may be more marked at the base than the apex.

Congenital affections of the other valves will create murmurs referable to the position of their orifices.

The diagnosis of transposition of the main vessels by cardiac physical signs is impossible. Transposition of the viscera may exist in connection with this anomaly, and may be recognised.

DIFFERENTIAL DIAGNOSIS.—There may be difficulty in deciding in some instances whether a cardiac murmur is of congenital or acquired origin.

No certain rules can be laid down, but the physician will be guided by the collateral signs, the past history of the patient, and the occurrence of any illness which would be likely to have laid the foundation of any cardiac disease. In the absence of any guidance from these records it may be noted that the murmurs of the common forms of malformation are systolic in time, that the murmur is not conducted in the manner usual in the acquired forms, and that it may have been observed in early childhood. In the more severe forms there would be evidence of much enlargement of the right ventricle, with probably some tendency to clubbing of the fingers. In the slight forms there would be no evidence of any secondary effects, or of mechanical interference with the heart's action.

DURATION OF LIFE.—There is considerable difference in the age attained in the various cases of cardiac malformation; the majority of those in whom there is any very serious defect do not survive birth more than a few days.

In some the mechanical difficulty of the circulation makes it impossible for life to be carried on for any great length of time; while in others with a considerable degree of malformation the circuit through the heart and great vessels is sufficiently free for life to be maintained for some years. Many persons with a slight degree of malformation, such as a patent foramen ovale, or a small aperture in the ventricular septum, have died at an advanced age, and have never presented any cardiac symptoms.

The duration of life in pulmonary stenosis depends partly on the degree of the obstruction, but more particularly on the condition of the cardiac septa. The prognosis is more favourable when there is some defect in the septum, as by this means relief is afforded to the overcharged right auricle and ventricle. In atresia of the pulmonary orifice life is much more abbreviated, and will also depend mainly upon free communication between the two sides through imperfect septa. In a few cases the patients have lived for some time when the lungs derived their supply from vessels supplied by the aorta.

In transposition of the main vessels the length of life is usually not great, but in some instances the patients have survived to adult life or even longer. An open condition of the septum, or patency of the ductus arteriosus, is favourable for the prolongation of life. With complete absence of the ventricular septum the majority die in infancy, but a few have survived to adult age.

The cause of death in a large number of infants is due to mechanical interference with the circulation. A considerable number die of convulsions, cerebral abscess, bronchitis, or pulmonary complaints. Those who live to adult age are peculiarly prone to pulmonary tubercle, and probably the great majority die from this complaint, or from cardiac failure. Dropsy is comparatively rare. A septic endocarditis is occasionally engrafted upon the malformed valves or stenosed orifice.

TREATMENT.—The treatment in congenital heart disease is mainly hygienic. The surface of the body must be carefully protected against cold, and a warm climate is desirable. Violent exertion or over-exercise is apt to produce palpitation and shortness of breath, and should be avoided.

A carefully regulated diet, especially in childhood and infancy, is of importance. Special precautions should be taken to prevent the onset of bronchial affections and convulsions, which are the commonest causes of death at an early age. The special liability to tuberculosis of those who reach adult age must not be forgotten. The treatment of any complications must be directed to the relief of the more urgent symptoms, and the remedies employed would be those which are applicable to similar conditions ensuing in the course of other heart affections.

The "Schott treatment" for the relief of the dilatation may perhaps be of benefit in suitable cases.

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L. H.

DISEASES OF THE PERICARDIUM

The Normal pericardium.—Before proceeding to discuss the morbid changes of the pericardium, a few remarks must be made concerning this structure in health. The pericardium is a fibro-serous sac, which surrounds the heart and the origin of the great vessels. It is of a somewhat conical shape, the base of the sac resting upon the diaphragm, and being connected with it; while its narrower portion is directed upwards. The external or fibrous layer is dense and unyielding; it is attached very firmly to the central tendon of the diaphragm, more loosely to its muscular structure, especially towards the left, by areolar tissue. The fibrous layer is continued for some distance along the large blood-vessels in the form of tubular prolongations, which become gradually lost upon and incorporated with their coats. The inferior vena cava passes through the floor of the pericardium to reach the heart, which is tethered to the sac by the attachment of the vessel to the foramen quadratum in the central tendon of the diaphragm.

The serous membrane lines the fibrous sac, and is reflected over the surface of the heart, thus constituting its parietal and visceral portions. These portions are continuous along the great vessels, about an inch to an inch and a half above the base of the heart; the aorta and pulmonary artery being enclosed in a common sheath, and a passage, named the "transverse sinus of the pericardium," being formed between these vessels and the auricles. The serous layer is also reflected on the superior vena cava and pulmonary veins, and forms a deep recess behind, between the entrance of the right and left veins into the left auricle. The inferior vena cava has a very scanty covering. A triangular fold—the "vestigial fold" of Marshall—formed by a duplicature of the serous layer enclosing areolar tissue and fat, with vessels and nerves, passes between the left pulmonary artery and the subjacent pulmonary veins. The pericardium has an abundant supply of vessels, lymphatics, and nerves, the last being derived from the phrenic, vagi, and sympathetic nerves.

Externally the pericardium is in contact anteriorly and laterally with the pleuræ covering the lungs; except below and in front where it approaches the surface in an angular space behind and to the left of the sternum, a space which varies in extent and shape in different instances. Under perfectly normal conditions the uncovered portion is somewhat triangular in outline, with the base below; it is bounded on the right by a line along the middle of the sternum from between the fourth cartilages, on the left by a line from the same point to the apex of the heart. The pericardium is attached by fibrous bands to the manubrium and ensiform cartilage. Behind it is in relation with the contents of the posterior mediastinum; and the structures to be more especially remembered on this aspect are the œsophagus, descending aorta, bifurcation of the trachea and left bronchus, and the other structures which form the root of the left lung. The phrenic nerves pass down, one on each side of the pericardium, on their way to the diaphragm.

In health the contiguous surfaces of the pericardium are kept moist by the usual secretion of serous membranes; this never collects in such quantity as to be capable of detection by physical examination during life, though at post-mortem examinations more or less fluid is always found in the sac, and it may amount to an ounce or two, or even more. Part of this, however, and in some cases most of it, has certainly exuded after death. The rubbing together of the surfaces during the cardiac movements, on account of their smoothness and moistness, does not give rise to any appreciable external sign.

The pericardium of an adult man with a healthy heart is capable of holding from fourteen to twenty-two ounces of fluid; that of a boy between six and nine years old, about six ounces when the sac is distended to the full by injecting water into it, by means of a syringe, through an opening made in the anterior part of the pericardium (Sibson). The late Dr. Begbie (10) gave the amount that could be injected in an adult as between twelve and eighteen ounces; but he stated further that the pericardium is distensible. The heart does not completely fill the sac, and is capable of some degree of movement within it.

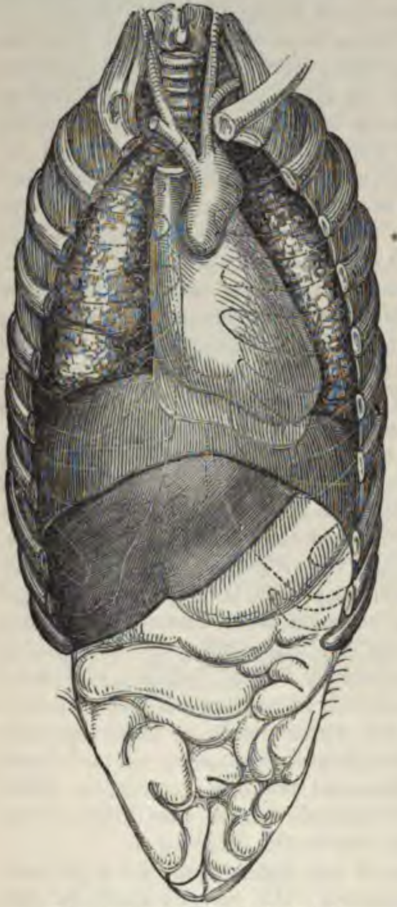

FIG. 83.—Pericardium not distended. (Sibson.)

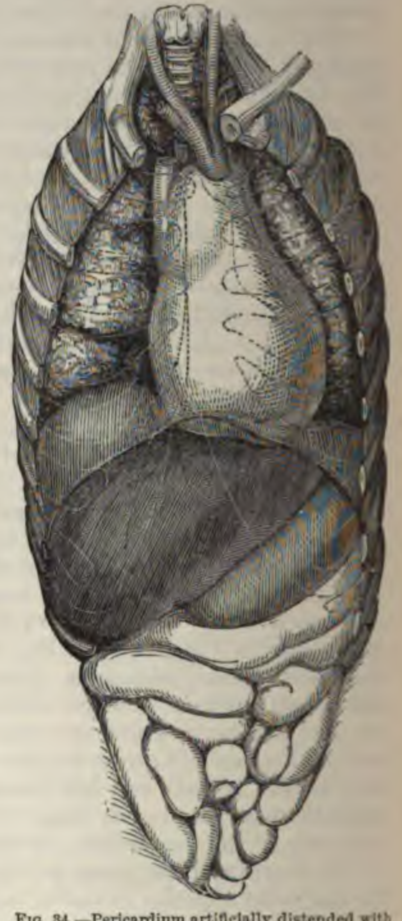

FIG. 84.—Pericardium artificially distended with fifteen ounces of fluid. (Sibson.)

MORBID CONDITIONS OF THE PERICARDIUM.—The pericardium is liable to certain very definite morbid changes; but, before discussing the more important of these, it will be convenient to refer briefly to certain conditions of this sac, which, although morbid, are in the large majority of instances more of pathological than of clinical interest or consequence, being indeed usually only revealed when a necropsy is made.

1. The pericardium in exceptional cases is the seat of more or less extensive *congenital defect*. In some instances this is very slight, and of no importance whatever; but there may be a fissure or opening in the sac of sufficient size to allow the heart to protrude through it, constituting a form of *ectopia cordis*. Rarely the defect is so considerable that the organ lies in the left pleural cavity, in contact with the lung, and covered only with the serous visceral layer of the pericardium; while the parietal portion is represented by fringe-like reduplications at the origin of the great vessels, or by "a kind of loose fold or pocket, which is found on the right side or upper part of the heart." "The defect seems to consist in the pericardium, which is apparently reflected from the external coat of the aorta, not being prolonged so as to cover the front of the heart and become attached to the diaphragm" (Peacock). Although under ordinary circumstances this last condition cannot be detected during life, it might certainly give rise to unusual and embarrassing signs should inflammation and effusion occur.

2. *Diverticula* or hernia-like pouches have been met with very rarely in connection with the pericardium. They are the result of pressure from within; usually by chronic pericardial effusion, exceptionally by blood. The fibrous layer becomes thinned or yields at a spot, and the serous lining protrudes as a sac, with a wider or narrower opening; it is generally of small size, but has been found sufficiently large to contain three to four ounces of fluid.

3. In the case of a greatly enlarged heart, where the pericardium is otherwise unaffected and free from obvious disease, this structure will of necessity become more or less *stretched* and *distended* in proportion to the size of the organ, and it may become *thinned* in the process. I am not aware that such a condition in itself gives rise to any discoverable signs or injurious consequences, but the condition may be assumed under such circumstances. An aneurysm of the heart walls, or of the intra-pericardial portion of the aorta, would also tend to push out the sac locally, and might even perforate it. Should pericardial effusion occur under such conditions the signs might be unusual.

4. At post-mortem examinations certain *white spots* or *patches* (*maculæ albidæ*) are frequently observed associated with the pericardium, the nature and origin of which have given rise to far more controversy than their importance demands. They are also known as *tendinous* and *milk-spots* (*maculæ v. insulæ tendineæ v. lacteæ*), and as "corns" or "callosities." At one time they were thought not to be pathological, but certainly they cannot be normal. The main discussion has turned on the question whether these spots or patches are or are not the result of inflammation. It cannot be doubted that the great majority of them are not of acute inflammatory origin at any rate; and the meanings attached to "chronic inflammation" by different pathologists are so totally at

variance, that it really does not matter whether we attribute them to such a process or not. My strong personal opinion is that these changes are almost always directly due to the constant mechanical attrition or irritation to which certain parts of the pericardium are subjected during the cardiac movements. They are met with in progressive frequency as age advances; it has been affirmed, indeed, that they do not occur in children at all; this statement is incorrect, but they are extremely rare in such subjects. They are decidedly more common in males than females, as might be anticipated if this view of their causation be correct; and also in persons in whom, from their occupation, much friction between the pericardial surfaces might be expected. Moreover, the white spots are by far most frequently observed on the visceral pericardium, over the portion of the front of the heart which, being uncovered by lung, comes chiefly into contact with the inner surface of the chest wall, that is to say the base or middle of the right ventricle; and they are not uncommon at the apex of the left ventricle. They do occur, however, on other parts of the surface; at the origin of the great vessels, as white stripes on the auricles, and along the course of the coronary arteries. They are met with very exceptionally on the parietal pericardium. Some of these changes are similar to those which affect other serous membranes, and cannot be very well explained; others are no doubt the remnants of a definite past pericarditis, when they present special characters, and are occasionally accompanied by adhesions or their remains in the form of filamentous fibrous bands: or there may have been a localised and trifling "dry" inflammation, which has not been detected during life.

Milk-spots are most common on large, hypertrophied and strongly acting hearts, but they are by no means confined to organs of this description. In character and structure they are whitish and more or less opaque, being in some cases of a dead white or pearly colour; they are generally circular in outline; of varying size, being usually about half an inch in diameter; and, as a rule, cannot be detached from the serous membrane, with which they seem to be intimately incorporated. Indeed they then consist merely of a local fibroid thickening or sclerosis of this structure, due to a hyperplasia of the connective tissue; rather perhaps to a condensation of fibres previously existing than to a development and increase of new fibres. Occasionally patches are met with presenting a smooth or granular surface, decidedly opaque, and of some degree of thickness and firmness, which can be peeled off from the underlying membrane, with which they are more or less loosely connected. Such patches are inflammatory in origin.

Clinically these conditions are generally regarded as of no consequence. Certainly they do not give rise to any cardiac symptoms whatever, and as a rule are not revealed during life by any signs. From personal observation, however, I feel sure that some white spots or patches on the pericardium are capable of originating a limited friction sound which, under certain circumstances, might be mistaken by an inexperienced or careless investigator for an early sign of acute pericarditis.

5. In rare instances what may be called *foreign bodies*, lying free in the pericardial sac, have been found at necropsies. Some of them have been soft and smooth, varying in size from a pea to a bean; others firm, fibrous, occasionally stratified, or calcified, either in a central nucleus or throughout—the so-called *cardiac calculi*. These bodies have been regarded as polypi detached from the inner surface of the pericardium; or as results of fibrinous or calcareous deposits about some foreign substance. They have never been diagnosed during life.

6. It may be mentioned, lastly, that, as a consequence of prolonged chronic pericarditis in extremely exceptional instances, the pericardium becomes the seat of extensive *calcareous deposit*, which may actually convert it into a complete *calcified shell* surrounding the heart; and the change may even encroach upon the cardiac walls, constituting the so-called "bony heart." Calcified spots or patches in connection with this sac are not uncommon. Although these conditions might be suspected under certain circumstances, it is very doubtful whether they can be demonstrated clinically; yet it has been affirmed that a calcified pericardium may give rise to a peculiar percussion sound of an osteal quality.

Having thus disposed of changes of the pericardium which are almost exclusively of pathological interest, I now proceed to deal with those diseases which are clinically important; and, taking a comprehensive survey, they may be indicated as follows:—I. Acute fibrinous and sero-fibrinous pericarditis. II. Suppurative pericarditis — Pyopericardium. III. Chronic pericarditis—Chronic effusion—Pericardial adhesions and thickening. IV. Hydropericardium—Dropsy of the pericardium. V. Hæmo- or hæmatopericardium—Blood in the pericardium. VI. Pneumopericardium and its effects—Gas in the pericardium. VII. New growths and parasites.

The diseases just enumerated are attended with pathological effects which give rise to well-recognised abnormal conditions, often of a very pronounced character. These conditions not only affect the pericardium and its contents, but also frequently influence neighbouring structures; while in most cases they are revealed clinically by well-marked and characteristic physical signs. It is very desirable at the outset to have a definite general knowledge of their nature, and of the signs to which they severally give rise. They may be comprehensively summed up as—(i.) abnormal states of the pericardial surfaces; (ii.) accumulations of fluid in the pericardial sac; (iii.) accumulations of gas, or of gas and fluid together; (iv.) pericardial adhesions of various kinds; (v.) thickening of the pericardium, usually associated with adhesions. It must be remembered that these abnormal physical conditions may be variously combined in particular cases. I now proceed to discuss the several diseases of the pericardium enumerated in the previous paragraph, and in the order there given.

I. ACUTE FIBRINOUS AND SERO-FIBRINOUS PERICARDITIS. ACUTE INFLAMMATION OF THE PERICARDIUM.

Acute inflammation and its results constitute by far the most frequent and important morbid conditions of the pericardium with which we have to deal in medical practice; and they often lead to serious consequences, both immediate and remote. As an acute affection pericarditis varies considerably in different cases, whether as regards its intensity and extent, the rapidity of its progress, the nature and amount of its pathological products, or its terminations and ultimate effects; but the complaint must always be looked upon with concern. In some instances it may be described as subacute rather than acute, but there is no line of demarcation between the two classes.

Etiology and Pathology.—In accordance with modern views of the relation of micro-organisms to acute inflammatory diseases, it may be assumed that particular agents of this nature are immediately concerned in originating acute pericarditis. It will be more convenient and practical, however, in the first instance, to point out the circumstances under which this affection occurs, and afterwards to try to ascertain what is really known of its bacteriology.

It has been customary, from an etiological point of view, to divide cases of acute pericarditis into *primary* or *idiopathic*, and *secondary*. The former include those which cannot be referred to any of the recognised causes of the disease, and in which it attacks an apparently healthy subject. Under such circumstances the complaint has been usually attributed to chill of some kind, but cases thus originating are probably of a rheumatic nature. In some cases called idiopathic the patients were drunkards, or were suffering from the effects of privation. In my own experience I have never met with an instance of acute pericarditis which, when carefully investigated, could not be included as a "secondary" event in one or other of the etiological groups now to be discussed.

(*a*) *Rheumatic pericarditis.*—This is by far the most important variety, and the number of cases coming under this head is very much larger than that of all other cases put together. There are several features to be noticed in this group, and in their discussion I propose to draw attention to some interesting and practical facts observed by the late Dr. Sibson, whose article on "Pericarditis," in Reynolds' *System of Medicine*, contains the outcome of extensive personal observations, with comments, many of which are noticed or quoted in the following pages. The definite connection between acute rheumatism and pericarditis has long been recognised. The pericardial inflammation is not to be looked upon as a mere complication, but is an essential part of the disease. The frequency of the association has been very differently stated by different writers, and doubtless it varies under different circumstances. In rheumatic cases pericarditis is not nearly so common as endocarditis; Sibson found the latter fully three times as frequent in those which he observed. Further, he noted that, in the large majority of cases of pericarditis, endocarditis was present also. The late Dr. Sturges drew special attention to this association in children, and he applied the names *peri-endocarditis* or *carditis* to the combination, which he regarded as exclusively rheumatic. He also laid down the proposition that "the rheumatic heart inflammation of children when pericardial is always endocardial as well, and when endocardial is extremely likely, with the recurrence of rheumatism, to involve the pericardium also." My own experience is fully in accord with the observations of these eminent physicians, and the effects of a combination of pericarditis and endocarditis come before us in a considerable proportion of the cases of chronic heart disease which can be traced to one or more rheumatic attacks in early life.

It has been stated on high authority that pericarditis of any kind is a rare disease in children; and the complaint has been said to prevail most frequently in middle life. More recent and accurate observations, however, have shown that such statements are quite contrary to fact. Sturges pointed out that pericarditis is very common in children. He noted that "out of 100 fatal cases of heart disease occurring at the Children's Hospital, Great Ormond Street, between June 1881 and April 1892, of which 54 were of rheumatic origin, and 46 due to other causes, in 6 only was there no evidence of pericarditis." When introducing the discussion on Acute Rheumatism at the meeting of the British Medical Association in 1895, Dr. Cheadle also spoke of pericarditis as less and less frequent with the advent of puberty. Certainly, so far as my own experience goes, while prepared to meet with rheumatic pericarditis at any age, it is in children, growing boys and girls, and young adults, that I have found it necessary to be more particularly on the look-out for the disease, and I am convinced that this is a point of considerable practical importance. [*Vide* art. "The Acute Rheumatism of Childhood," vol. iii. p. 44.]

Rheumatic pericarditis is decidedly more common in males than females, but the exact difference cannot be stated. Sibson found the proportion to be 1 in 4 to 1 in 6; and he explained this difference in part by the influence of age and occupation on acute rheumatism and its complications. He observed that one-half of the males and more than one-half of the females were below the age of 21; while two-fifths of the male and only one-fifth of the female patients were above the age of 25. Servants formed fully two-thirds of the female patients affected with pericarditis; and three-fourths of the servants attacked with pericarditis and endocarditis were below the age of 21. These facts Sibson explained by the hard occupation of patients of this class, in view of their time of life and constitution, which exposes them to the causes of acute rheumatism and its attendant inflammation of the heart. Those of tender age who followed no occupation were not attacked with inflammation of the heart with anything like the same frequency as young female servants. Women who at mature age followed occupations as laborious as that of the young servants were affected with pericarditis in but a moderate proportion, and in a comparatively mild form. He concludes—"We thus see, in brief, that in acute rheumatism affecting the female sex, youth with labour is nearly always attacked or threatened with endocarditis or pericarditis, or both; that youth without labour is thus attacked with comparative infrequency; and that mature age with labour is attacked much less frequently and much less severely with inflammation of the heart than youth with labour."

With regard to males Sibson observed the following facts:—Of laborious workers out of doors attacked with pericarditis only 1 in 10 was below the age of 21; while of indoor workers thus affected fully three-fourths were below that age. The scale was entirely reversed in those of older age. Of those labouring out of doors four-fifths were above 25; while of those working indoors only one-sixth were above that age. Sibson writes: "We here, I consider, find the explanation of the twofold fact, that the male cases of pericarditis usually combined with endocarditis outnumber the female cases by one-fifth, and that the number of the men so affected above the age of 25 is three times as large as that of the women. I think we may infer that excessive labour in men of mature age is a frequent cause of acute rheumatism having a strong tendency to pericarditis." He further concludes that "in both sexes the same causes produce, under like conditions, the same effects; and that a very large proportion of the young persons who work on foot indoors during many hours daily are attacked with inflammation of the heart when affected with acute rheumatism, while a very small proportion are thus attacked of the men and women of mature age who are engaged in the same manner."

The relation between the severity of the rheumatic condition, as seen in the joints, and acute pericarditis must next be considered. Sibson noted that in servants attacked with pericarditis the severity of the joint affection in the great majority of cases bore a strict relation to the severity of the heart affection. Taking all cases into account, however, this rule is by no means true in a considerable proportion of them, and it is highly important to remember that pericarditis may set in and become very pronounced while the articular complaint is comparatively or actually mild; and it may even occur alone, or come first of the rheumatic series. This statement applies particularly to children, who are liable even to carditis of rheumatic origin with little or no joint affection or pyrexia. Another observation made by Sibson is that in about half the cases, when the pericardial effusion is at its height, the joint affection tends to lessen in severity. The number of articulations involved, and the implication of particular joints, bear no relation to the frequency of pericarditis. The disputed question whether it is more prone to occur during first or subsequent attacks of acute rheumatism is not of much practical significance, though the general experience is in favour of first attacks. The appearance of the affection must be watched for during each attack, whether it has or has not previously occurred, unless indeed it has left behind universal adhesion.

Sibson stated that previous rheumatic attacks favour the occurrence of endocarditis much more than of pericarditis, and this is in accordance with my own experience. As to the time at which acute pericarditis supervenes in the course of a rheumatic attack, it seems to appear in a certain proportion of cases—according to Sibson, about one-eighth—at the very beginning, and to be coincident with the joint affection; or, as already stated, it may even precede such a manifestation. Not uncommonly it appears between the third and the sixth day; and, according to Dr. George Balfour, most cases occur within the first week of the rheumatic onset. In nearly one-half of Sibson's cases signs of pericarditis were observed on or before the eleventh day of the illness. On the other hand, the complaint may not be revealed for two or three weeks or more; and in seven out of sixty-three of Sibson's cases it showed itself between the twenty-fifth and sixty-third day. Moreover, it may follow a relapse of articular rheumatism, the pericardium having been quite free from any affection during the primary attack. In the case of children pericarditis may arise at any stage of the rheumatic series, but, according to Cheadle, most often it comes late, in association with recurrent endocarditis, when the heart is already hypertrophied and dilated (*vide* vol. iii. p. 38).

The opinion has been advanced that excessive action of the heart, set up by the rheumatic condition, may help in the production of acute pericarditis. This was evidently Sibson's opinion concerning the relative severity of the joint affection and that of pericarditis, for he writes: "When the joint affection is severe, it may call forth excessive labour or even tumultuous action of the heart. In acute rheumatism inflammation attacks the fibrous structures, especially if these structures are unduly strained, and the increased action of the heart may therefore, I consider, induce inflammation of the fibrous tissues of that organ, such inflammation being proportioned in severity to the augmented action of the heart."

Cold, damp, and changeable climate and season have necessarily a marked influence upon the frequency of cases of rheumatic pericarditis.

(*b*) *Renal pericarditis.*—The association of acute pericarditis with different varieties of Bright's disease cannot be doubted, but statistics seem to show that the frequency of this form depends upon certain circumstances. Sibson collected a large number of cases, from various sources, of renal disease thus classified, and found that pericarditis occurred in 8·1 per cent; but it differed materially in frequency in different countries, the proportion in Germany being 10·4, in France only 3, and in England 8·4 per cent. With regard to its relative frequency in the several varieties of Bright's disease, it appears from Sibson's statistics to be uncommon in connection with acute scarlatinal nephritis in young subjects, but frequent in adults who suffer from acute Bright's disease, as well as during the transitional stage to the large white kidney. When the latter has become established, however, the tendency to general pericarditis disappears almost entirely; yet it may occur in a partial or circumscribed form. In connection with the contracted granular kidney the complaint is far more common; and it is of average or moderate frequency in cases of lardaceous disease. It has also been stated to be especially associated with fatty kidney. Dr. George Balfour regards acute pericarditis as usually a late phenomenon in renal disease, and states that it is often a precursor of fatal uræmia. Sibson believed that over-action of the heart increases the tendency to pericarditis in Bright's disease, as well as the enlargement of the organ associated with the granular kidney. He also affirmed that great enlargement tends to develop partial into general pericarditis.

(c) *Pericarditis from extension or irritation.*—The occurrence of pericarditis as the result of extension of the inflammatory process from neighbouring structures is an important fact; and my observation of cases in which the disease has thus originated has led me to believe that they are more frequent than is generally known. In most instances it follows pneumonia or pleurisy, more particularly when the inflammation is on the left side. It spreads from the pleura to the outer surface of the pericardium, and thence penetrates to the interior of the sac, kindling a more or less pronounced pericarditis. Professor Shattuck has recently called attention to the frequency of the association of pericarditis with pneumonia. It must be noted, however, that in some cases in which these combinations of acute inflammatory diseases are met with in the chest, the pericardium was involved first, and from it the inflammation spread to other structures; or the whole of them may be implicated so rapidly that it is difficult or impossible to determine where the inflammation started. In exceptional instances the process may extend, through the diaphragm, from the peritoneum to the pericardium, without any direct communication between the two cavities. It will suffice to mention that cases are now and then met with in which pericardial inflammation is set up by some neighbouring cause of irritation, apart from the inflammatory diseases just considered; such as abscess, aneurysm, enlarged glands or tumours, or bone disease.

(d) *Traumatic and perforative pericarditis.*—These two forms may be considered together. They are of very rare occurrence, and I cannot speak of them from personal knowledge. The chief injuries from without which may cause pericarditis are a blow or contusion over the precordial region; fractured ribs; penetrating wounds by sharp instruments or gunshot wounds, and lesions produced by way of the œsophagus, especially by foreign bodies, purposely or accidentally swallowed, which may actually perforate the pericardium, or even gain access into its cavity, or, remaining lodged in the gullet, may injure the adjacent pericardium: examples of such bodies are false teeth, needles, or fish-bones. Perforative pericarditis may result from the bursting of any neighbouring abscess into the sac, whether associated with the bronchial glands, bone disease, or other like conditions; or, in very exceptional instances, a communication may be established from an empyema, from a phthisical cavity, or from the œsophagus if it be the seat of ulceration or new growth. Still more rarely the contents of an abdominal abscess find their way through the diaphragm into the pericardium; and even a gastric ulcer has perforated its walls. In all these cases definitely irritating or septic materials of different kinds gain access to the pericardial sac.

(*e*) *Pericarditis secondary to cardiac or aortic disease.*—A separate group may be recognised of cases in which acute pericarditis is secondary to some affection of the heart itself, or of the arch of the aorta. As already pointed out, this affection and endocarditis frequently occur together in rheumatic cases. It seems probable, moreover, that inflammation primarily involving the endocardium may traverse the myocardium, so as ultimately to implicate the pericardium as well; and this applies particularly to infectious endocarditis. Myocarditis itself, especially if ending in suppuration, may likewise originate pericarditis, quite apart from the bursting of an abscess of the walls of the heart into the sac. Among very rare causes may be mentioned cardiac aneurysm, or intra-pericardial aortic aneurysm. With regard to chronic diseases of the heart, pericarditis has now and then appeared in cases of valvular affection, chiefly aortic, especially when associated with cardiac hypertrophy; but the connection between these conditions is not very clear, and it seems to me that on careful investigation of such cases some more definite cause of the pericardial inflammation would probably be found.

(*f*) *Pericarditis associated with new growths.*—The chief kinds under this head are malignant and tuberculous pericarditis. The former class of growths may be situated in the heart, the pericardium, or both; and no practical distinction can be drawn between them. Tuberculous pericarditis is probably more frequent than is usually supposed, and should not be forgotten. When the inflammation is set up in chronic phthisis, apart from the bursting of a vomica into the sac, it is essentially a slow process; but in acute tuberculosis or very active phthisis it may certainly be acute. In very exceptional cases pericarditis seems to be the main tuberculous manifestation, and it is then rather subacute in its onset and mode of progress.

(*g*) *Septic pericarditis.*—This variety deserves separate recognition, as it may arise in all kinds of general septicæmia and pyæmia; though under such circumstances the pericardium is far less frequently affected than the pleura. Septicæmia associated with puerperal conditions and acute necrosis of bone have to be especially remembered in this connection.

(*h*) *Pericarditis associated with miscellaneous general diseases and blood-states.*—It will suffice under this heading to draw attention to the fact that in exceptional instances acute pericarditis occurs as a complication of some of the acute specific diseases, particularly scarlatina (most commonly during the period of desquamation, when it has been attributed to rheumatism or renal disease), measles, and small-pox; rarely of enteric fever, typhus, diphtheria, erysipelas, cholera, severe malarial fevers, scurvy, purpura, and hæmophilia; in these last conditions it is probably secondary, in some cases at any rate, to pericardial hæmorrhage: it may occur exceptionally in the gouty state; and in diabetes.

Bacteriology of acute pericarditis.—On a comprehensive survey of the etiological groups just considered, the relations of micro-organisms to the inflammatory process can in many instances be clearly recognised on general grounds. Such organisms have also been frequently demonstrated in the inflammatory products and in the pericardium itself. At the same time it cannot be said, so far as present researches go, that they are in any way specifically related to the disease. The notion seems to be gaining ground that the rheumatic process is due to a specific bacillus, to the direct action of which the pericarditis would in this case be attributable; but I am informed on the highest authority that up to the present time no such organism has been demonstrated. The organisms usually found are those ordinarily associated with the inflammatory process, namely, different kinds of streptococci and staphylococci. In cases following pneumonia the pneumococcus may be present, and it has also been found independently. Tubercle bacilli have been demonstrated in tuberculous pericarditis.

Morbid anatomy.—The changes which occur during the progress of acute pericarditis are similar in their general nature to those which characterise inflammation of other serous membranes. It is customary to describe the disease as following successively the stages of—(i.) Increased vascularity; (ii.) Fibrinous exudation; (iii.) Fluid effusion; (iv.) Absorption; and (v.) Adhesion. These stages, however, cannot always be definitely recognised, and in many instances they run more or less concurrently. Moreover, the pericardium itself is often involved in the inflammatory process. It will be expedient, in the first place, to describe individually the changes which take place during the progress of a pronounced case of acute pericarditis; and afterwards to point out the more important aspects under which they are presented in practice.

(i.) Hyperæmia, or increased vascularity, no doubt constitutes the earliest change in acute pericarditis. It involves the serous lining of the sac and the subserous tissue, and is accompanied with more or less parenchymatous swelling of the membrane. In its lesser degrees the hyperæmia is revealed by a fine network of vessels; but in its more pronounced form the surface is extensively and uniformly red, the redness being either bright or dark. Sibson described the appearance as follows:—"When the surface of the heart becomes inflamed, a blush of fine vessels, consisting of a velvety network, appears on the surface of the organ, and especially over the larger coronary vessels at the base and septum of the ventricles. The inner surface of the pericardial sac, wherever it rests upon the inflamed heart, kindles also into a blush of fine vessels. The surfaces of the heart and sac, instead of being smooth and glistening, become dull and velvety." Sometimes minute hæmorrhages are observed, especially around newly-formed vessels. The hyperæmic condition is of short duration, it may last but a few hours, and then either subsides or is concealed by exudation. As a matter of fact it is seldom seen at necropsies, and usually only in pericarditis connected with Bright's disease. It is said to be generally less marked in elderly and cachectic persons.

(ii.) The deposit of fibrinous exudation or inflammatory lymph is an invariable accompaniment of acute pericarditis; though its quantity, extent, mode of arrangement, and exact characters vary much in different cases. As a rule, it is observed both over the surface of the heart and the interior of the pericardial sac. In some instances there are merely a few shreds about the roots of the great vessels; in others a thin film or coating forms at different spots, especially on the visceral surface; or a more or less thick and stratified layer covers both surfaces extensively or universally, and is often very abundant. Owing to the incessant movements of the heart the arrangement of the exudation is often peculiar. It very rarely presents a smooth surface; and in the large majority of cases it exhibits an alveolar, reticular, or honeycomb pattern. Sibson thus well describes the appearance:—"Where the two surfaces touch each other, the soft lymph is drawn into threads and little pointed ridges and prominences, and wrought into a network, so that when ridges and prominences are present on the heart, ridges or prominences are present on the inner surface of the pouch lying upon it, and when a network of lymph covers the heart, a network of lymph lines the corresponding sac. The constant play of expansion and contraction of the heart alternately stretches and relaxes its coating of lymph, so that its surface resembles a honeycomb in structure." Laennec's well-known and oft-quoted comparison likens the appearance to that presented on suddenly separating two smooth pieces of wood between which a small pat of butter has been forcibly compressed. It has also been called the "bread-and-butter like" appearance; or it has been likened to tripe. It must be noted, however, that the lymph does not always present this kind of arrangement; it may exhibit a shaggy or villous surface, or peculiar characters, to which such names as *cor hirsutum*, *cor tomentosum*, etc., have been applied. When abundant, it is said to accumulate in large masses in the auriculo-ventricular groove and about the auricles. Should there be much lymph associated with fluid its surface is covered with floating shaggy processes, which sometimes have a mammillated aspect. Occasionally fibrinous papillæ or bands pass across between the opposing surfaces of the pericardium, and these may even form partitions.

The lymph exuded in pericarditis is usually of a whitish yellow, yellowish, or reddish colour; but it may be brownish. In a very short time a fine network of vessels is developed in its substance, and not uncommonly spots of hæmorrhage are present, or the whole exudation may be deeply stained. In connection with purpura, scurvy, and allied blood diseases alternating layers of blood and lymph are now and then observed. In consistence the material is, as a rule, somewhat firm and elastic, but it may present different degrees of softness down to that of an almost liquid jelly. Not infrequently it is mixed with serous fluid. In exceptional instances of a low type it has been described as granular, crumbling, or boggy. At first the exudation can be readily separated and peeled off from the surface of the membrane, but after a while it becomes more adherent and difficult to detach. In structure it consists of coagulated fibrin and cell elements, the latter chiefly occupying the deeper layers. When the material is very soft the cells are in great abundance, and at the same time molecular disintegration has taken place. Micro-organisms of different kinds may be found in the exudation.

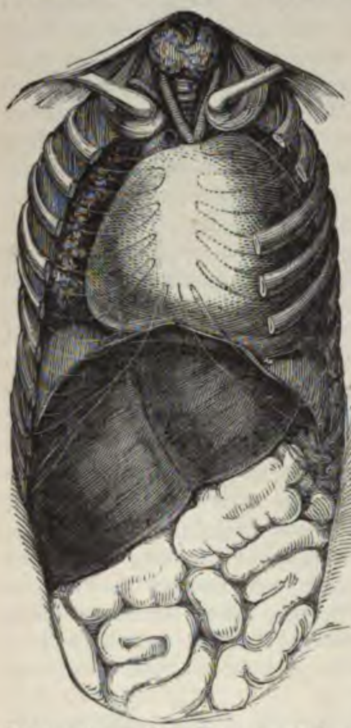

FIG. 35.—Case of pericarditis in which sac contained 3¼ lbs. of fluid. Patient under care of Sir James Anderson. (Sibson.)

(iii.) There can be no doubt that in not a few cases of acute pericarditis there is little or no fluid effusion; a form of "dry pericarditis" being met with, which can be recognised clinically. In such cases very rapid adhesion may take place between the visceral and parietal surfaces, even over an extensive area; the lymph being thick, sticky, gelatinous, and specially agglutinative. Sturges drew particular attention to this course of events in children. Occasionally a kind of network of fibrinous strings passes between the adjacent surfaces, the meshes of which are filled with serum. As a rule, however, during the progress of an attack of acute pericarditis, where there are no adhesions, an effusion of fluid takes place into the pericardial sac, separating its parietal and visceral layers. Effusion may indeed supervene after the formation of early soft adhesions, sometimes limited to one side; or when the sac is partially filled with heavy gelatinous masses of lymph. The average quantity of fluid is from 8 to 12 ounces, but it may range from an ounce or two to two or three pints or more—Balfour says "several pints." The amount of fluid effusion is by no means in proportion to that of the fibrinous exudation, and the result of the inflammatory process may chiefly be evidenced by either one or the other. It is in rheumatic pericarditis that large accumulations usually occur, and the effusion then generally collects and increases rapidly, often reaching its acme in two, three, or four days (Sibson). In opposition to this statement Dr. John Broadbent affirms that "it is the exception rather than the rule to find effusion of any extent in cases of pericarditis of rheumatic origin." Dr. Church seems to be of the same opinion [*vide* "Acute Rheumatism," vol. iii. p. 16]; and Dr. Cheadle states that in children the effusion, though fluctuating in amount, is never very large, and is usually reabsorbed quickly. In my experience, cases of rheumatic pericarditis have differed very much in the quantity of effusion. In

Bright's disease the quantity is often very small. Abundant effusion is likely to be met with in scorbutic cases, and in these as much as five pints has been recorded.

The effusion in acute pericarditis is generally of a serous or serofibrinous character, and yellowish or greenish in colour; it is most commonly bright, clear, and transparent; but may present small fibrinous particles or flakes in suspension, or be opalescent, or even more or less cloudy and opaque. Occasionally it is brownish or reddish. The specific gravity averages about 1018. Under certain circumstances, as when pericarditis is associated with purpura or scurvy, the effusion is obviously mixed with more or less blood or its colouring matter—"hæmorrhagic pericarditis." The cases in which the inflammation leads to the formation of pus will be separately discussed; and it will suffice to state further that in very rare instances—of which there is an obvious explanation in the presence of some general septicæmic condition, the effects of neighbouring gangrene or malignant disease, or the entrance of air containing septic micro-organisms into the sac—its contents undergo a putrefactive change, and become "ichorous," foul in appearance and odour, or actually stinking.

It will be convenient in the present connection to discuss briefly from a general point of view the immediate effects of pericardial effusion upon the sac itself and its contents, as well as upon neighbouring structures, effects which are met with by far most frequently in cases of acute pericarditis; and at the same time to point out the changes which the inflammatory process is apt to set up in the pericardium and heart, and which tend more or less to influence and modify these effects. Obviously they must vary considerably in nature and degree, according to the amount of the fluid accumulation, and the rapidity of its collection. The fact must be acknowledged that a certain quantity of effusion is sometimes found in the pericardium at the autopsy, it may be as much as 6 or 8 ounces, which had not given rise to any evident disturbance, and was not detected during life. In all such cases, however, which have come under my personal observation, there has been every reason to believe that the effusion had taken place shortly before death, from obvious causes, and usually under circumstances rendering adequate physical examination impracticable; and no doubt it is often increased by transudation of serum from the vessels after death.

Beginning with the pericardium itself, when a collection of fluid exceeds a certain quantity the sac necessarily becomes more and more distended, in proportion to its amount, and at the same time stretched and thinned, so far as the normally tough and firm parietal pericardium will permit. When acute pericarditis has lasted for some time, however, and the structures forming the walls of the sac itself are involved in the inflammatory process, they become more or less swollen, thickened, soft, and yielding; so that the pericardium becomes capable of far greater distension than in its natural state. As the fluid accumulates in increasing quantity the sac undergoes changes in form, which have been well described and figured by Sibson. When artificially distended with 15 ounces of fluid, he noted that the pericardium became pyramidal or pear-shaped, and in more detailed description he writes: "It is formed, so to speak, of a larger and a smaller sphere, the smaller one resting on the top of the larger. The larger and lower sphere contains the heart, the ascending vena cava, and the pulmonary veins; and the smaller sphere holds the great vessels. The distended sac occupies the whole centre of the chest, filling up the space between the sternum in front and the spinal column behind, and extending across the chest from a little within the right nipple to a little beyond the left nipple. The whole sac is lengthened; its smaller end reaches upwards almost to the top of the sternum; and its floor, being formed by the central tendon of the diaphragm, presents a large spherical prominence that bulges downwards into the abdomen, occupies the epigastrium, and reaches as low as the tip of the ensiform cartilage and the lower edge of the sixth costal cartilage." This description will apply to the shape which the pericardium usually assumes when distended with fluid from pathological causes; but when, owing to inflammatory changes in the walls previously referred to, they give way further, the form alters considerably. "As the sac cannot expand to a material degree either upwards towards the neck, or downwards towards the abdomen, it yields sideways and backwards, and widens to the right and especially to the left" (Sibson). Under these circumstances, in short, its width becomes decidedly disproportionate to its height, and it loses its pyramidal outline, becoming in extreme cases almost globular.

The secondary changes of the heart and great vessels which may supervene in acute pericarditis, as well as the effects of a considerable effusion of any kind on these structures, must be discussed in some detail; and in individual cases they should always be borne in mind, and carefully studied from a clinical point of view. On these matters there are important differences of opinion, and they have given rise to much controversy.

There can be no doubt that inflammatory changes beginning in the pericardium are apt to extend to the muscular tissue of the heart itself, and this tissue may also undergo an acute degenerative change. These lesions are, speaking generally, proportionate to the intensity and duration of the pericarditis; but they are most frequently met with in the hæmorrhagic and purulent varieties. They are naturally more pronounced in the superficial layers, but may gradually extend throughout the entire thickness of the walls. The degenerative changes sometimes attain a high degree, even in wholly acute cases. Whether the inflammatory process may extend from the pericardium through the cardiac walls to the endocardium is difficult to determine positively, but I think it is highly probable. The changes in the heart tissues have been attributed, not only to extension of the inflammation, but also to persistent high bodily temperature, and to the circulation of toxins in the blood. Degeneration has, moreover, been supposed to result from the inflammatory products pressing upon the coronary arteries, and thus impeding the normal distribution of blood to the cardiac walls. Pericardial effusion, however abundant, has no direct influence upon the structure of the muscular tissue of the heart. It must be noted that the nerves distributed to the surface of the heart and great vessels may be involved in the inflammatory process.

Dr. John Broadbent maintains that considerable dilatation of the heart, especially of the right ventricle, occurs during pericarditis; and he has brought forward cases to prove that the physical signs usually attributed to pericardial effusion are really due to this condition. That such a dilatation does take place in some instances is indisputable, especially when extensive adhesions have rapidly formed; but my experience is certainly opposed to the supposition that it is usually an immediate result of pericarditis, or that it is apt under these circumstances to be mistaken for effusion.

What is the mode in which the fluid collects, and what position does the heart assume within the sac? These questions have been the subject of special controversy; and although to some writers they present no difficulty, and are unhesitatingly answered in a particular way, without reserve, I must confess that in my own clinical experience of individual instances I have not always found them easy of solution. It will be convenient at the outset to explain Sibson's later views on this subject, and some of his remarks deserve to be quoted at length. Describing the mode in which fluid collects in the pericardium, he writes: "At first it falls into the back part of the sac, but as it increases in quantity it makes a space for itself between the floor of the pericardium, which it depresses, and the lower surface of the heart, which it elevates; . . . and the result of this is to displace the apex and body of the organ and its great arteries upwards and forwards." He adds: "The heart, elevated by the fluid between the under surface of the ventricles and the base of the pericardium to a degree proportioned to the amount of the fluid, leaves the broader part of the chest below, and ascends into the narrower part of the chest above." In another place he writes: "The distension of the pericardium with fluid produces two other effects on the heart. (*a*) The heart is heavier than the fluid in which it plays, and its ventricles consequently tend to sink backwards so that the left ventricle rests upon the posterior wall of the pericardium. (*b*) The other effect of pericardial distension on the heart is the lifting or tilting upwards of the organ within the sac. The heart is attached by its great vessels to the posterior and upper part of the sac, and the whole organ therefore tends to shrink upwards and gravitate backwards towards its points of attachment." Sibson concluded that the natural effect of this gravitation, shrinking, and upward displacement of the heart, owing to great accumulation of fluid in the sac, if not modified by other agencies, would be to cause the interposition of a layer of fluid between the front of the heart and the anterior wall of the chest. He affirmed, however, that in practice this is not usually the case over the mass of the ventricles, though a layer of fluid covers the lower part of the right ventricle.

The displacement of the apex of the heart upwards and outwards in cases of pericardial effusion was formerly taught as an indisputable fact. By most authors at the present day, however, though not by all, this doctrine is regarded as a mistake. The general opinion is that the fluid collects towards the front, and that the heart, being heavier than the fluid, falls or sinks backwards, away from the anterior thoracic wall; the ventricles, right auricle, and great vessels being successively covered from below upwards, and thus separated from the parietal pericardium. Some writers have maintained that an effusion first collects about the base, which is turned downwards, the heart lying rather more horizontal than normal, and the apex turned outwards; but this part is described as descending when the diaphragm is pushed down by the effusion.

Another opinion is that the position of the heart is not altered. This is the opinion of Dr. William Ewart (19), who affirms that the apex will be found in the usual situation at any necropsy on a case of uncomplicated pericardial effusion; and that whilst the heart has preserved its normal situation the floor and the sides of the pericardium have receded from it. Dr. Ewart regards the impossibility of any elevation of the apex as almost self-evident. He writes: "Slight mechanical displacement might conceivably be brought about by one circumstance only—the lifting by the distended pericardial sac of the tracheal bifurcation and of the bronchi, and with them of the pulmonary veins and of the heart. Practically this rise is very inconsiderable, and moreover it does not directly influence the ventricle. On the other hand, we must remember that the heart is tethered to the bottom of the pericardium by the attachment of the inferior vena cava to the foramen quadratum in the central tendon, and that the considerable descent of the diaphragm must depress the level of the right auricle and tend to depress the apex, far from allowing it to rise. I have in some cases detected a lowering of the heart's apex in pericardial effusion, and with it a more median position of the heart, which then tends to hang more vertically from the aortic arch, the latter becoming slightly straightened."

The late Dr. Sturges, in summing up the opinions just discussed, expressed his belief that "though apparently conflicting, they are in fact reconcilable. They all express the truth in various circumstances. The heart may be moved either forwards, upwards, or backwards in effusion; or it may remain where it was; and of the factors that determine its conduct, pericardial adhesion, here or there, temporary or permanent, is the chief." He further stated: "I have repeatedly in fatal cases of pericardial effusion inserted needles, just before the post-mortem examination, into the proper apex place, and above the fifth right costal cartilage, close to the sternum, without being able to detect upon opening the chest any dislocation of the heart. The validity of such experiments may be questioned; but there are clinical facts to show that the early pushing forward of the heart, . . . although it may be the rule, is not without exception. The fluid may cover the heart from the first." It appears to me that Dr. Sturges' observations are rational and practical; and in dealing with particular cases it is well not to have too fixed or positive an opinion as to the position of the heart in pericardial effusion. Should the sac be quite free, there can be no doubt that in very abundant effusions the organ is covered by the fluid; and this is evident at necropsies under such circumstances, the body being in the usual recumbent position.

The next question is what effects, if any, are produced by pericardial effusion upon the walls of the heart and great vessels, when it becomes so considerable as to interfere directly with these structures? Sibson writes on this point: "The muscular walls of the ventricles are so thick, and their action is so powerful, that the direct effects of the fluid pressure upon them cannot be very great. But the pressure of the fluid tells inwards upon the weak and unresisting walls of the auricles, the vena cava descendens within the pericardium, and the pulmonary veins, so as to compress and lessen the cubic contents of those vessels and the auricles, and to resist and impede the currents of blood, on the one hand from the system along the cava, and on the other from the lungs along the pulmonary veins. This partial blocking of the double stream from the system and the lungs to the heart lessens the contents of the organ, and tends to diminish the size of its cavities. At the same time the supply of blood to the aorta is lessened, and the ascending aorta is therefore also compressed by the fluid. The pulmonary artery, however, owing to the obstacle to the flow of blood through the lungs, tends to resist the pressure of the fluid in the swollen sac, and to remain distended." This seems to be a correct description of the case of large effusions. Sibson, however, was further of opinion that in cases of pericarditis the compressing influence of pericardial effusion is counteracted by the protecting and sustaining covering of lymph, which to some extent shields the weaker parts of the heart, and strengthens the naturally feeble walls of the auricles and veins.

As regards the effects of pericardial effusion upon the action of the heart, it is believed that the systole of the auricles and ventricles is not restrained by such a collection; indeed, according to Traube, the systolic motion of the organ is greater than normal, the fluid being less resistant than the pericardium. The compression of the walls already referred to may, however, interfere with the diastolic distension, and thus diminish the flow of blood into the cavities, especially into the auricles. The direct interference with the entrance of the blood from the veins into the auricles, and impairment of the normal elastic traction of the lungs upon the walls of the heart, add to this difficulty.

It will be obvious that distension of the pericardium with fluid must interfere with neighbouring structures in proportion to its amount, and such consequences are chiefly seen in the respiratory apparatus. Some observers maintain that the portions of the lungs in front of the sac are pressed at first against the inner surface of the anterior wall of the chest. The ordinary effects of pericardial effusion upon these organs are complex. It necessarily embarrasses them more or less, and large collections of fluid also press upon the bifurcation of the trachea and the bronchi, especially the left bronchus. Hence it is found in many cases that the upper lobes of the lungs, particularly the right, are abnormally distended with air, or in a state of inflation, and in time become the seat of catarrh also; while other portions are collapsed in various degrees. As the effusion increases, and attains an excessive amount, it pushes these structures to either side and backwards, at the same time compressing them more and more, the left lung especially, which in extreme cases may become almost or even completely collapsed and airless. In some instances rapid and repeated serous effusion has taken place into one or both pleuræ in connection with great pericardial distension. Ewart states that pleuritic effusion is among the most common complications of severe pericardial effusion; that it frequently begins in the right pleura, but not uncommonly occurs ultimately on both sides; but that its occurrence belongs to the later rather than to the earlier stages. This condition is regarded as of mechanical origin, being attributed to pressure on the vessels in the roots of the lungs.

A very abundant pericardial effusion may press upon the œsophagus and descending aorta sufficiently to interfere with their channels. Whether the phrenic or other nerves within the thorax may be affected by the mere physical consequences of such an accumulation it is difficult to say; but some observers are of opinion that this may be the case, and it is highly probable, especially if the effusion be rapid.

A considerable pericardial effusion will tend to cause more or less protrusion of the corresponding portion of the thoracic walls, particularly in young subjects. When these walls have become rigid no such protrusion can take place. In a downward direction the diaphragm is not only embarrassed, but often considerably depressed, as well as the contiguous viscera, as chiefly evidenced by the liver.

(iv.) The course of events and the ultimate pathological results in acute pericarditis differ much in different cases. The natural tendency is for any serous or sero-fibrinous effusion to become absorbed sooner or later; sometimes very rapidly. According to Sibson's observations in rheumatic pericarditis the fluid after reaching its acme soon begins to lessen, and in from four to six days usually falls to the normal amount. There is every reason to believe, moreover, that even fibrinous exudation, up to a certain amount, can be absorbed completely, after undergoing a molecular fatty change; a little pericardial thickening or opacity at the most being left behind. The probability of such absorption is in inverse ratio to the extent and thickness of the lymph deposited, and to the duration of the inflammation. In respect of the "white spots" on the pericardium, it may be well to note again that fibrous patches resulting from pericarditis are usually distinguished by greater thickness and extent, irregular distribution, and special characters; and as a rule by the coexistence of adhesions. Very rarely irregular knob-like projections or pedunculated outgrowths are formed, and the latter may even become detached, and lie loose in the pericardial sac.

(v.) In most cases, after absorption of the fluid, or where only lymph has been exuded, adhesions of various kinds and degrees are formed. At first these are soft and easily broken down, and on account of the movements of the heart firm and permanent adhesions are much less easily established than in other serous membranes. Loose adhesions of connective tissue are probably torn by the repeated pulling and stretching; and it is believed that the cardiac action considerably interferes with the circulation in the newly-formed vessels. On this question Sibson writes: "In most instances slight threads of adhesion form between the sac and portions of the right auricle, and often also between the sac and the apex and interventricular septum, that being the portion of the front of the heart that presents the least movement during the action of the ventricles. These soft threads of adhesion are generally drawn out by the oscillating movements of the heart, until they at length yield and break away, but sometimes permanent adhesions form which may be partial or universal."

I have a strong impression that there is a general tendency to make light of the conditions remaining after acute pericarditis, or at any rate not to regard them as of much consequence; and I feel it necessary, therefore, to call special attention to the fact that well-marked pericardial adhesions not uncommonly persist, particularly in young subjects, and subsequently often become of decided importance. A new growth of connective tissue takes place, originating mainly in the cells present in the exudation; the fibrinous portion taking no part in the process, but being absorbed after undergoing fatty degeneration. In severe cases the tissues of the pericardium itself contribute to the growth. As the subject of pericardial adhesions is separately discussed in this article no further reference need be made to it here. It must be noted that in exceptional cases an ordinary inflammatory effusion into the pericardium does not undergo absorption, but remains as a chronic collection, or may become hæmorrhagic or purulent. These conditions will also be referred to more fully hereafter.

According to the extent of the disease, cases of pericarditis have been divided into *circumscribed* or *local*, and *diffuse*, the latter being in many instances *general* or practically universal. Local pericarditis may be met with in any part, but is chiefly observed at the base, about the origin of the great vessels; and the inflammation may thence extend to the coats of the arteries, so far as they are covered by pericardium, and subsequently give rise to thickenings and callosities.

In the preceding discussion pericarditis has been dealt with only so far as it affects the sac internally. It must be mentioned, however, that in not a few instances the external surface of the pericardium is acutely involved at the same time, or alone; though it is more commonly involved in a chronic process. The condition has received the names of *external pericarditis*, *mediastino-pericarditis*, or *pleuro-pericarditis* when the contiguous surfaces of the pleura and pericardium are affected. This form of disease and its results will be more conveniently dealt with later, and in other connections.

Clinical history.—Acute pericarditis presents considerable differences in its clinical history, depending upon a variety of circumstances; and this fact must be clearly recognised at the outset, and always borne in mind in practice. At the same time the phenomena to be watched for and studied are definite, and when at all pronounced bear an obvious relation to the morbid changes which are associated with the disease. The signs observed by physical examination are of special clinical value, for the symptoms are not uncommonly anything but characteristic; while the more important of these signs can be investigated as a rule without much difficulty, and it is only by their aid that we can positively determine the pathological conditions of the pericardium. Indeed, it must never be forgotten that, when symptoms are practically absent or latent, they may reveal the presence of even serious acute pericarditis; and this statement applies still more to cases in which the inflammation is localised. Moreover, physical examination gives the only trustworthy information as to the progress of the morbid changes.

Taking a comprehensive survey of the circumstances under which acute pericarditis usually supervenes, it might be anticipated that an attack is not ushered in, as a rule, by any striking premonitory symptoms, such as rigors and the like; and experience confirms this conclusion. The fact must not be overlooked, however, that in certain classes of cases the illness may begin with phenomena of this nature; nor must it be forgotten that even rheumatic pericarditis may appear as a primary acute disease, before the joints or any other structures reveal the presence of the rheumatic condition.

Discussion of symptoms.—From what has been just stated, it may be gathered that it is useless to attempt to give a definite clinical picture of acute pericarditis, and it will be more practical in the first instance to consider individually the several symptoms which may be associated with this disease; remembering that they differ much in their exact nature, severity, and combinations in particular cases.

(i.) *Subjective sensations.*—Pain is a symptom to be looked for in the early stage of acute pericarditis; but it is by no means always present, nor does it bear any necessary proportion to the seriousness of the attack. I can corroborate, from personal observation, the statement that severe pain may be associated with a limited dry pericarditis of short duration; while, on the other hand, it is a familiar fact that in cases of large effusion no such sensation may have been complained of from first to last, or it may have been so slight and transient as not to have attracted any attention. In young children pain seems to be generally absent. Sibson made numerous careful and interesting observations on this symptom in rheumatic pericarditis, and some of his conclusions are incorporated in the following remarks. In the majority of cases where pain is present it is referred to the precordial region, extending usually from the right of the sternum at its lower two-thirds to the left nipple. This pain is more or less continuous, but varies in severity, being in exceptional instances very intense. In character it is described in different cases as dull, aching, shooting, stabbing, burning, or tearing. Sibson noted that it came on, as a rule, at an early stage, afterwards diminishing; and usually relief, which was permanent, came when the effusion was at its height. Pain may, however, either precede or follow friction sound. Occasionally a return of the pain occurs with a relapse. The suffering is often increased by deep pressure or percussion; and now and then there is tenderness without spontaneous pain. Sibson observed in many cases that the skin over the region of the pericardium was tender and sensitive; so much so in some instances as to forbid the slightest manipulation of the chest, and to make a full examination of the heart impossible. Sometimes this superficial hyperæsthesia is certainly very remarkable. In other cases the structures of the intercostal spaces seem to be tender.

Another not uncommon seat of pain or tenderness, or both, is the epigastric region, where, according to some writers, it is even more frequent than in the precordial. The tenderness is said to be most marked at one or other of the costal angles, and is particularly brought out when upward pressure is made. Epigastric pain comes on, as a rule, later than that over the heart, and in a considerable proportion of Sibson's cases it appeared when the effusion was at its height. Both varieties are likely to be increased by the act of respiration and by bodily movements. Sometimes painful sensations radiate in different directions from the central points. A deep pain in the chest, between the shoulder-blades, was noticed in a few cases by Sibson; it was increased by swallowing or eructation, and occasionally was only thus brought out. He thought that in these instances the pain was seated in the back of the inflamed pericardium; and he also believed that pain and fulness after food might result from pressure made by the distended stomach over the lower and posterior part of the sac. In exceptional instances pain of an anginal character, shooting up the left side of the neck, to the ear, to the shoulder, or down the arm, is associated with acute pericarditis; but endocarditis has almost always been present at the same time, and generally chronic valvular disease also. The sensations just discussed are believed to be located mainly in the sentient nerves distributed to the surface of the heart, the pericardial sac itself and the portion of diaphragm incorporated with it, or the pleura covering the pericardium. They are often associated together in different combinations. Moreover, there may be pain in one or other side, evidently of pleuritic origin, or referred indefinitely to the chest. Taking all his cases together, Sibson found that there was pain of some kind over the heart or pericardium in 70 per cent.

Other subjective sensations besides those actually painful are not uncommonly complained of in acute pericarditis, as the disease progresses; and especially if a large accumulation of fluid takes place. They are described in different cases as feelings of precordial uneasiness, oppression or pressure, a weight or load over the heart, tightness, or ill-defined distress and anxiety. The respiration may be distressed; and not only may the patient be conscious of the disturbed heart, but sometimes there is a distinctly painful form of palpitation. Baumler has noticed painful sensibility of the left side of the larynx, increased by every movement of the heart.

(ii.) *Disorders of the cardiac action and pulse.*—It might naturally be expected that acute pericarditis would affect the action of the heart in various ways. In the early stage the heart is excited and irritable, as evidenced by increased rapidity and force of the beats, the movements in some instances being more or less tumultuous. Subsequently, not only as the result of large effusion, but also of the implication of the myocardium and its nerves, as well as of other influences, the cardiac action becomes more or less embarrassed and ineffectual, and this may culminate in marked feebleness or exhaustion, with irregularity and intermittence, or even faintness or actual syncope which, in exceptional instances, has come on suddenly or very rapidly, and proved fatal. With regard to the frequency of the pulse, according to Sibson, "it rises in number as the disease rises in intensity, is at its greatest rapidity when the disease is at its acme, and falls in number as the disease declines." "During the early stage the pulse usually mounts up to 90, 100, or even 120; but later on it tends to become more rapid, and in rare cases reached 160." It may, however, not be much changed from the normal, or from what it was before the pericarditis supervened; or after an initial acceleration it may soon subside. In exceptional instances the pulse is retarded in the course of the disease. A much quickened pulse-rate, 120 or 130, without adequate rise of temperature, is said by Dr. Cheadle to be very characteristic of the subacute pericarditis of early life. In the early stage the pulse is generally full and strong, and may be increased in tension; as the case progresses it becomes small, weak, often dicrotic, and of very low tension. Dr. Ewart, however, draws special attention to the large and slapping pulse which he has frequently observed in pericardial effusion. He writes: "The peculiarity of the pulse is its great size and velocity of impact, and the sudden collapse of the wave. In fact it is Corrigan's pulse, almost of a typical kind, though never so extreme as in well-marked aortic regurgitation." Irregularity or intermittence may accompany a similar disturbance of the cardiac rhythm; occasionally this is an early phenomenon, but usually comes on later. It has been stated that in some cases of copious pericardial effusion the left carotid and radial arteries are smaller and pulsate less forcibly than the corresponding arteries on the right side (Traube). The sphygmograph has been much used to investigate the pulse in cases of acute pericarditis, but I venture to doubt whether it is of much practical value. Speaking from personal experience of this disease, I think it must be acknowledged that no definite description of the pulse can be given; but at the same time the study of it in individual instances affords most useful information, and it needs to be watched at frequent intervals. In grave cases it may become almost imperceptible. The *pulsus paradoxus* has been observed occasionally in large pericardial effusions.

(iii.) *Respiratory system.*—Some disturbance of breathing is noticed in the great majority of cases of acute pericarditis, varying much in its degree and exact characters, but often well marked or even decidedly grave. By pain in the early period respiration is rendered quick and hurried, but restrained and shallow; and this cause may also modify the movements later, when the physical effects of pericardial effusion, as well as other influences, especially the cardiac changes, come into play. If there be much fluid, actual dyspnœa supervenes, the respirations increasing in frequency, with marked activity of upper costal breathing, but more on the right side than the left. As it accumulates, the breathing becomes more and more difficult and laboured, the alæ nasi work, the extraordinary muscles are called into play, there is a corresponding sense of oppression, distress and air-hunger, and the patient may have to be propped up more or less. In extreme cases the dyspnœa is very urgent, the respiratory movements are greatly impeded, and there is persistent orthopnœa, or the patient instinctively bends forwards to seek relief. As a rule it is more comfortable to lie on the left than the right side, but dorsal decumbency is usually preferred. Occasionally the dyspnœa is intensified paroxysmally. As the fluid is absorbed the respirations fall, and the breathing improves; but a relapse may cause fresh disturbance. With regard to the pulse-respiration ratio, even at the early period it may be altered from the normal to 3:1; and later the proportion may come to be 2 or 2½ : 1. The difficulty of breathing interferes with the act of speaking; and changes in the voice have been described in exceptional instances by Sibson and others, and attributed mainly to pressure upon or implication of one or both recurrent nerves. A short, irritable, spasmodic cough is not uncommon with a large pericardial effusion, and there may be a little mucous frothy expectoration. Distressing and painful hiccup is an occasional symptom, attributed to implication of the phrenic nerve in the inflammatory process.

(iv.) *Dysphagia.*—Difficulty or pain in swallowing is occasionally noticed, mainly the result of the pressure of a large pericardial effusion upon the œsophagus; but sometimes it appears to be due to nerve-irritation. Deglutition is more difficult in the recumbent posture, and is made easier by raising the shoulders and bending forwards. In exceptional cases the difficulty is only associated with swallowing solids, or is brought on by œsophageal spasm induced by an attempt to drink. Rarely a feeling of spasmodic choking in the throat or along the gullet is complained of.

(v.) *General symptoms and appearance.*—More or less pyrexia may be expected in cases of acute pericarditis, but it does not present any special course or characters. Its manifestation in rheumatic cases may not be attended with any increase of temperature previously raised; it seldom rises above 102° or 103° at any time, and may soon subside. Sometimes it is practically normal throughout, or only slightly elevated, from 99° to 100° or 101°, especially in children. It is affirmed that rapid absorption of inflammatory products may occasion some rise of temperature. As a rule, strength is fairly maintained; but in some instances, especially in children, there is marked prostration. In severe cases of acute pericarditis, especially when associated with endocarditis, the expression generally indicates anxiety, distress, or depression; and the face is flushed, dusky, or pallid, or presents alternating hues. Rarely it has a muddy or glazed appearance. The eyes at the same time are dull, heavy and injected. Sibson noted a marked change in the appearance of the patient in four-fifths of his cases, to which he attached much importance: as the complaint subsided he found that the aspect quickly improved, the eyes becoming bright and clear, the cheeks rosy, and the expression often quite suddenly cheerful. This observer attributed the flushing and pallor of the face to the influence of the inflammation on the nerves at the surface of the heart, inducing reflex dilatation or contraction of the arteries of the head and face. He stated that the flushing seemed to tint the face all at once.

The most striking general symptoms in the grave forms of acute pericarditis are those indicative of interference with the aeration of the blood, and of general venous obstruction. The patient then presents a more or less livid or cyanotic appearance; sweating, often profuse; fulness of the veins of the neck, sometimes with pulsation; and in extreme cases coldness of the extremities. Possibly dropsy of the legs may occur. A large effusion in children is said to affect the action of the heart more rapidly than in adults, and to lead to an earlier interference with the circulation. In these subjects progressive anæmia and wasting are in some instances pronounced symptoms. Perspiration was observed by Sibson to be usually copious when there was flushing. The amount and characters of the urine will depend very much upon the condition with which the pericarditis is associated. It tends to be deficient in quantity, and to present the usual changes associated with the rheumatic and febrile states. Albuminuria may occur altogether independent of renal disease.

(vi.) *Nervous symptoms.*—Patients suffering from pronounced acute pericarditis are generally very restless, but movements may be checked by the rheumatism. Headache and sleeplessness are frequent symptoms, and slight delirium is not uncommon. Vomiting is sometimes a marked symptom in acute pericarditis, and is regarded as of nervous origin. In exceptional cases nervous disturbances become very prominent, and may be grave, such as delirium, either active and noisy, or even violent and maniacal, chiefly nocturnal; or low and muttering: sometimes a transition from one to the other variety takes place. The condition may resemble delirium tremens, the patient being strange in manner, excited, and incoherent; or there may be a tendency to stupor, semi-unconsciousness, temporary insensibility, or actual coma; or to motor disorders, such as subsultus tendinum and jactitation, "risus sardonicus," clonic or tonic spasms, rolling of the head from side to side, choreiform movements, general convulsions ending in extreme exhaustion, tetanic rigidity; or to curious emotional attacks in early life, in which the child is moved to tears or laughter by a word (Cheadle); or even to temporary insanity, usually with taciturn melancholy, and often with hallucinations; this derangement may last some time, but is ultimately recovered from. The particular symptoms of this class and their combinations differ much in different cases, and delirium may pass into coma. They cannot, as a rule, be referred directly to the pericarditis, but depend rather on the disease to which it is secondary, its associated complications—hyperpyrexia in some instances, the state of the nervous system, want of oxygenation of the blood, the previous habits of the patient, or other circumstances. Some authorities, however, attribute the phenomena to the influence of the pericarditis upon the nervous system; and Bright believed that such an influence can be communicated through the phrenic nerve to the spinal cord, and is the cause of choreic and tetaniform affections. Dr. George Balfour writes: "The occurrence of delirium in the course of rheumatic fever ought at once to direct attention to the heart; and the sudden occurrence of spasms or coma in chronic renal disease is only too frequently found to be associated with pericarditis, both of these phenomena being probably caused by the saturation of the blood with the products of retrograde metamorphosis, due to the sudden development of the inflammation." It is important to note, however, that even in the gravest cases of acute pericarditis, ending fatally, and accompanied with other intrathoracic inflammations, there may be no marked nervous symptoms throughout, the patient being perfectly clear to the last.

Sturges described a very fatal form of pericarditis in children, with little or no effusion, exudation being followed by rapid adhesion; and he referred the grave symptoms observed in these cases to the nervous system. Dr. Cheadle also calls attention to occasional cases in such subjects which run an acute course with dangerous symptoms; but he states that, as a rule, they arise when pericarditis occurs late, when the heart is already seriously damaged by previous attacks of endo- or pericarditis, and when the secondary changes of dilatation and hypertrophy, and perhaps adherent pericardium, have already advanced to a marked extent (*vide* vol. iii. p. 45).

Physical signs.—In discussing the physical signs of acute pericarditis, it is convenient to recognise certain stages corresponding to the progress of the morbid changes already described; although it must be clearly understood that there is no actual line of demarcation between them, the conditions which give rise to these signs being commonly present at the same time. It may be remarked that the excited or turbulent action of the heart which often occurs at the onset of the disease will be evident on examination, but there is nothing characteristic in this disturbance.

First stage.—During the early period the signs to be looked for are those indicative of abnormal states of the contiguous pericardial surfaces, which are pressed and rubbed against each other during the movements of the heart. They are commonly known as *pericardial friction-fremitus* or *thrill*, and *friction murmurs* or *sounds;* signs which must be considered in some detail. Many deny that any phenomenon of this kind can be produced by mere increased vascularity and dryness of the surfaces, but in my opinion a faint friction murmur may certainly be thus originated. It is, however, to the fibrinous exudation that the more pronounced and characteristic signs of the early stage of acute pericarditis are due. They can, in my opinion, be brought out only when the conditions producing them exist on the anterior aspect of the heart, although some writers have made a contrary statement; and it is highly probable that when the inflammatory lymph is of a very soft consistence, it may not give any definite sign perceptible on physical examination.

(i.) *Pericardial friction-fremitus or thrill.*—The tactile sensation thus named is practically only recognisable in a comparatively small proportion of cases of acute pericarditis, and when present it is always accompanied with a loud friction-sound. For the detection of this sign careful palpation with the finger-tips may be needed, and I believe that it can thus be made out more frequently than is generally supposed. It depends more immediately upon the amount and characters of the exudation, though it is also influenced materially by the force of the heart's action.

When any abnormal sensation is felt over the precordial region, the chief point to be determined is whether it is a pericardial fremitus or an endocardial thrill. It must suffice to summarise here the more characteristic features of a pericardial fremitus, and to any one practically acquainted with the usual endocardial thrills the points of difference between them will be at once apparent.

(*a*) A pericardial friction-fremitus has no definite "focus of intensity" (Sibson), and varies much in its seat and extent. As a rule its area is circumscribed, and it is felt more towards the base of the heart or over the middle of the precordium; sometimes it is limited to the apex. Now and then, however, the sensation is perceptible over a considerable extent of surface, or in more than one spot. (*b*) It always gives the impression of being peculiarly superficial, as if the condition producing it were close under the finger. (*c*) The rhythm is practically systolic, the fremitus being associated with the cardiac impulse; it usually begins and ends rather abruptly, and there is no shock at the close: sometimes it is irregular in rhythm, differing in exact time in successive beats. (*d*) In quality a pericardial friction-fremitus gives more or less the impression of the rubbing together of rough surfaces, and in different cases it is described as harsh and grating, rasping, vibrating, or creaking. Sibson speaks of it as being in many instances a sensation of a thrill, but in my experience the feeling has certainly never been like that of an endocardial thrill. (*e*) As a rule this sign is short-lived and transient (Sibson), and, should it last any time, often changes from day to day in its situation, extent, and characters. Pericardial friction-fremitus may be simulated by one of pleuritic or mediastinal origin, brought out by the movements of the heart; and this fact must not be forgotten.

(ii.) *Pericardial murmur or friction-sound.*—It is by the adventitious sounds heard on auscultation that, in the large majority of cases, the early stage of acute pericarditis is recognised. Some writers distinguish between a pericardial murmur and friction-sound, and Sibson made a great point of this distinction; but there is no practical line of demarcation between them. In the following remarks, therefore, I shall employ the term "pericardial friction-sound" inclusively, merely remarking that the so-called murmur may be regarded as representing the minor degrees of this sign, and that now and then an adventitious sound of pericardial origin may no doubt closely resemble an endocardial murmur in quality.

It is requisite to have a comprehensive and intelligent conception of the more characteristic features of pericardial friction-sounds, so as to be able to contrast them with those of endocardial murmurs; but as a rule they are easily distinguished. Moreover, by careful attention to the special qualities of the sounds heard, it is practicable in many cases to arrive at a tolerably definite notion of the conditions of the pericardium upon which they depend. It may be observed here that pericardial friction-sound also may unquestionably be simulated by one of pleuritic origin, or by a sound originating in the mediastinal cellular tissue over the pericardium.

(*a*) While usually more or less circumscribed in extent, pericardial friction-sound does not correspond in any way, as regards its situation or its point of maximum intensity, to any of the recognised endocardial murmurs. In some cases it is audible extensively, though not of the same loudness throughout its area; but even then it is generally defined with remarkable abruptness, and is never conducted in the directions peculiar to the several intra-cardiac murmurs; nor, according to my experience, can it ever be heard over the back of the chest. During the early stage of acute pericarditis friction-sound never extends beyond the region of the heart, but in the later it may do so in exceptional instances. When associated with a fremitus it usually spreads, as from a focus, in all directions more or less beyond the area where this sensation can be felt.

(*b*) As a rule pericardial friction-sound has a double or to-and-fro rhythm, being both systolic and diastolic; but in some instances, or over certain parts of the heart, it may be confined to the systole. In pronounced cases the two parts are of about equal duration, each sound seeming to fill up its respective space, leaving a short interval of silence between the two (Sibson). They may, however, occupy the whole time of the cardiac movement, thus often giving at first a confused impression to the ear; but intermissions can be made out. As regards the cardiac sounds, the pericardial murmur seldom corresponds exactly in rhythm with either, and is prolonged beyond them, while they are often distinctly audible through it; though, on the other hand, the friction-sound may be so loud as to drown them entirely. Moreover, its precise time is frequently irregular, varying with successive beats of the heart. This is more especially noticed in connection with the diastolic portion, which is usually not so loud as the systolic. A double "to-and-fro" adventitious sound heard in connection with the cardiac movements, of maximum intensity at the same spot, is regarded as highly characteristic of pericardial origin. It has been stated that four murmurs may be audible, the two sides of the heart each producing a systolic and diastolic murmur of different duration; but that most frequently three are heard, one presystolic, belonging to the systole of the auricles, and two longer sounds, corresponding to the systole and diastole of the ventricles. Rarely pericardial friction is divided into several parts.

(*c*) While varying much in its intensity, pericardial friction-sound strikes the ear as being peculiarly superficial; and this character is more pronounced in proportion to its loudness. Sibson spoke of it as a "surface noise."

(*d*) The precise characters of a pericardial friction-sound vary considerably within well-recognised limits, according to the nature of the conditions upon which it depends. In the large majority of cases it conveys to the ear a distinct impression of the rubbing together of contiguous surfaces during the cardiac movements; in short, it is of the quality of a "friction-sound." In its lesser degrees it is soft or grazing, whiffing, brushing, or rustling; but its more pronounced varieties are described by such terms as harsh, rough, grating or vibrating, and creaking, like the bending of new leather. Bouillaud classified pericardial friction-sounds as grazing, new leather sound, and grating, which are the common varieties. Sometimes they resemble the rubbing of sand-paper. Under certain circumstances the sound is more of a crackling (as of paper or parchment), clicking, churning, or rumbling character, or it may be scraping, scratching, or sawing. It has also been described as "sticky." While thus various, pericardial sounds are as a rule entirely different in quality from endocardial murmurs. Moreover, the double pericardial friction-sounds never begin with an accent or shock, but begin, continue, and end, as a rule, with the same tone throughout (Sibson). When pericardial friction-sounds and endocardial murmurs exist together, the combinations may be very peculiar and difficult to define.

(*e*) *Tests.*—In certain cases in which a pericardial friction-sound is not distinctly audible, but its presence is suspected, or where it is doubtful whether an adventitious sound heard on auscultation be pericardial, endocardial, or pleuritic, the difficulty may be cleared up by the judicious application of certain recognised tests. These may also help in affording a more correct knowledge of the conditions of the pericardial surfaces upon which a friction-sound depends.

(*a*) *Pressure test.*—Firm but not too forcible pressure with the stethoscope over different parts of the region of the heart has long been known as an important and useful test of pericardial friction-sound. It may bring out this sign when not previously audible, especially over the lower two-thirds of the sternum (Sibson). Its effect upon the sound, when present, may be to intensify it and make it louder; to enlarge the area over which it is heard; to modify its duration and rhythm, rendering it more prolonged and continuous, or making it double—systolic and diastolic—when previously only systolic; to alter its character, tone, and pitch, causing it to become more harsh and rough, and especially grating or creaking, or these qualities come out more prominently under pressure (Sibson described a peculiar double sound thus elicited, like the noise made by sharpening a scythe); or to silence the natural cardiac sounds previously heard, or even mask endocardial murmurs.

(β) *Respiration test.*—The act of respiration may unquestionably produce a definite influence upon pericardial friction-sound, especially as regards its extent, less frequently as to its intensity and quality; and possibly some help in diagnosis may thus be afforded in doubtful cases. It is generally stated that inspiration always increases pericardial friction-sound. Sibson observed that the area of the friction-sound increased below during inspiration in a large number of cases; while in a much smaller number it increased above during expiration. It became more loud or harsh sometimes during expiration, sometimes during inspiration; and in one instance it disappeared at the end of a deep breath. I may refer here to pleuritic friction simulating pericardial. As a rule it can be distinguished by its situation at the left border of the pericardium, and by its cessation when breathing is stopped, but certainly not always.

(γ) *Effects of exertion and posture.*—Should a pericardial friction-sound not be heard at all or but feebly, in consequence of weak action of the heart, it might possibly be brought out or made louder by exciting the organ by some kind of effort. Moreover, it certainly may be intensified or increased in area by bending the body forwards; while occasionally it is audible in the recumbent but not in the sitting posture. Change of position may affect the locality and extent of this sign in certain cases. Personally I doubt whether the tests mentioned under this head are of much practical value, and at any rate special discretion and caution are required in carrying them out.

(δ) *Variability.*—Marked changes in the site, rhythm, intensity, and characters of pericardial friction-sound from day to day, or within shorter periods, constitute a series of most important tests in a large number of instances.

Stage of effusion.—When fluid collects in the pericardial sac in any quantity, it may be expected to give rise to a very definite group of physical signs, varying in their degree according to its amount and other circumstances. It must not be forgotten, however, that rapid adhesion may take place without any effusion, so that the phenomena of this stage may be entirely wanting, especially in children. Conversely, it occasionally happens that a large quantity of fluid accumulates very rapidly and insidiously without forerunning friction signs, or at any rate without their detection. The possibility of considerable cardiac dilatation, as described by Dr. John Broadbent, must also be borne in mind, lest a wrong diagnosis of pericardial effusion be made.

We shall first consider how pericardial effusion may modify the friction phenomena. Sibson made numerous observations on this problem, and his conclusions were believed by him to support his own views of the position of the heart in these cases. According to his observations the tendency of the effusion is to shift the whole region of actual friction, and with it the friction-sound, upwards; and steadily to increase its area in this direction and to the right and left. In the large majority of cases he found the area of friction-sound greater at the time of the acme of the effusion than before; in a few it remained the same; in two only was it less than before. In two instances the friction-sound disappeared during the acme, but Sibson attributed this mainly to lowered heart power. He observed that the tendency is for the sign to increase in intensity also, but in this respect the exceptions were more frequent. It may be stated with certainty that even large effusions do not necessarily obliterate the friction-phenomena; indeed there may be an abundance of fluid, at least as much as two pints, in the pericardium, while these signs are pronounced. Dr. George Balfour goes so far as to affirm that if a friction-sound be once heard over the base of the heart in front, no amount of subsequent effusion suffices to efface it. I do not think that this statement will hold good absolutely, and friction-sound over other parts of the heart is likely to be completely silenced as a rising tide of fluid separates the two pericardial surfaces.

I proceed now to discuss the more positive signs which are associated in various degrees with pericardial effusion.

1. The tendency of pericardial effusion, when in sufficient quantity, is to cause proportionate bulging or prominence of the corresponding portion of the front of the chest, and occasionally this is a very striking sign. Some writers have asserted that this condition leads to a uniform enlargement of the left side; but although there may be a certain degree of general distension the prominence is always greater in front. In the case of a large effusion the margin of the sternum and the left costal cartilages are pushed forwards, while the ribs are raised bodily upwards, and the intercostal spaces widened. In extreme instances the fulness may extend from the second to the sixth or seventh cartilages, but chiefly from the fourth to the sixth; the spaces are sometimes felt to be quite smooth, and an obscure sense of fluctuation may possibly be detected in them. Sibson stated that in very large effusion the dorsal portion of the spinal column deepens itself and is curved backwards. Bulging is naturally more easily produced in children and growing subjects, on account of the yielding condition of the chest walls; while it may be entirely prevented by rigidity of these walls, which thus adds seriously to internal embarrassments by the fluid. The enlargement has been partly attributed by some writers to inflammatory paralysis of the intercostal muscles.

Dr. William Ewart (19) regards what he calls the "first rib sign" as important in the diagnosis of considerable pericardial effusion. This is an altered relation between the left clavicle and the first rib, so that the upper edge of the latter can be felt as far as its sternal attachment. He writes: "This points to a raising of the clavicle not only in its outer but also in its inner portion, and to a relaxation of the ligament between it and the first rib. The left clavicle is apparently lifted to a higher level than it is possible for the first rib to reach."

A prominence of the epigastric region may be noticed in cases of abundant pericardial effusion, due partly to the fluid itself pressing down the diaphragm, partly to the liver, which is also depressed and congested.

In his case Dr. Samuel West observed the rare phenomenon of a peculiar elastic semi-fluctuating depression in the epigastrium, which he regards as additional evidence of effusion having its seat in the pericardium. Dr. Clifford Allbutt has met with a similar phenomenon.

2. Certain signs of pericardial effusion, associated with the cardiac movements, as revealed by the impulse and apex-beat, demand careful study:—

(*a*) There can be no doubt that one of the obvious effects of a free and uncomplicated accumulation of fluid in the pericardium is a real or apparent elevation of the apex-beat, which seems at the same time to be carried somewhat towards the left. Moreover, the movement becomes unusually extensive in an upward direction, its diffusion being often easy to recognise by inspection and palpation. According to Sibson's observations there is, as a rule, a relation between the extent of the effusion and the height of the impulse. This he found raised so that its lower boundary corresponded to the fourth or even the third space or cartilage, being also felt at or to the left of the nipple line. The diffusion varied according to the position of the lower boundary; but in the large majority of cases, at the time of the acme, it extended above this boundary to the extent of one or more of the higher intercostal spaces. In exceptional cases the impulse was diffused from the fourth to the second spaces, but generally it was confined to the fourth and third, or the third and second spaces. Sibson attributed the raising and diffusion of the impulse to elevation of the heart by the fluid, and to enlargement of the right ventricle and pulmonary artery from obstruction of the flow of blood through the lungs. He believed, in common with other authors, that it is the actual apex-beat which is felt, displaced upwards and to the left. At the present time, however, most writers regard this opinion as erroneous, and consider that the impulse is communicated by a higher portion of the heart. Thus Dr. George Balfour believes that the true apex is pushed inwards by the effusion, and removed from the anterior wall; while the part of the heart actually in contact with the chest wall is nearer the base of the ventricles. Dr. William Ewart (19) also writes: "That an impulse can usually be felt there (at the third space) is not surprising, since the antero-posterior diameter of the chest at that level (between sternum and spine) is not much greater than that of the heart itself, whilst the left lung no longer intervenes between the latter and the chest wall. The impulse is not, however, that of the apex of the heart, but rather of its base." My personal observations lead me to agree with these views on the whole, but there may be conditions in certain cases to cause actual uplifting of the apex-beat.

Series of figures (Nos. 36 to 43), from cases described by Sibson, illustrating the morbid conditions in pericarditis and the physical signs associated therewith. The black spaces correspond to the pericardial dulness, the curved lines to the impulses, and the zigzags to the friction-sounds. In Fig. 41 there is complete adhesion of the pericardium to the heart.

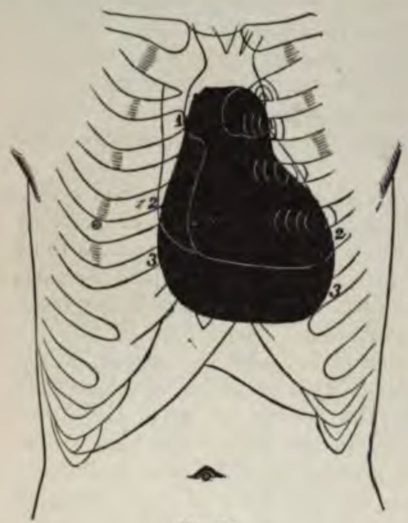

FIG. 36.

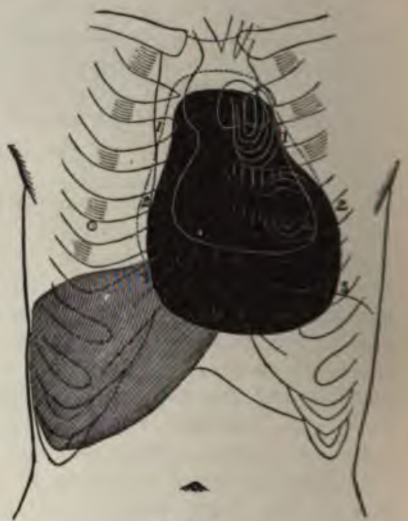

FIG. 37.

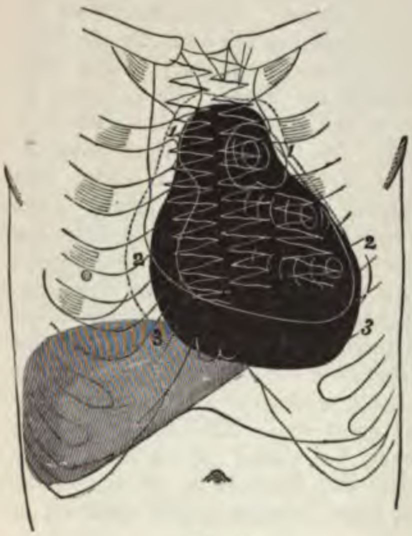

FIG. 38.

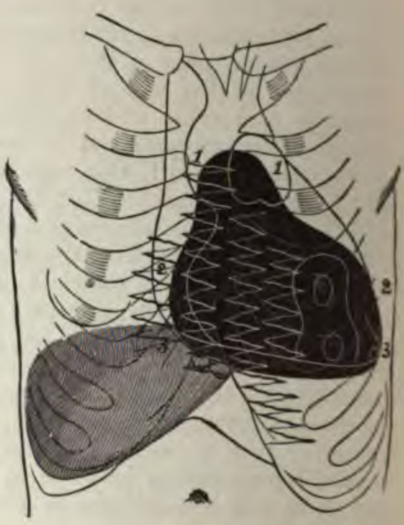

FIG. 39.

Occasionally it has been noticed in acute pericarditis with effusion that the apex-beat is somewhat lower than normal. This may be due to enlargement of the heart; but it has also been attributed to the presence

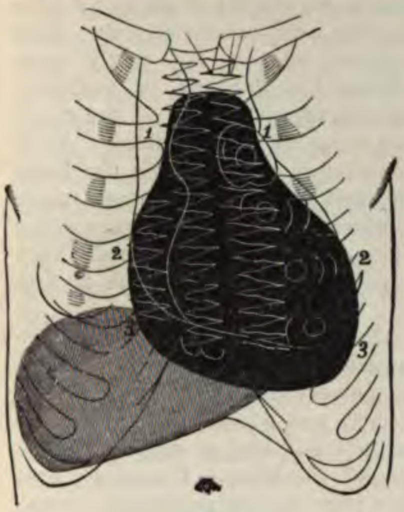

FIG. 40.

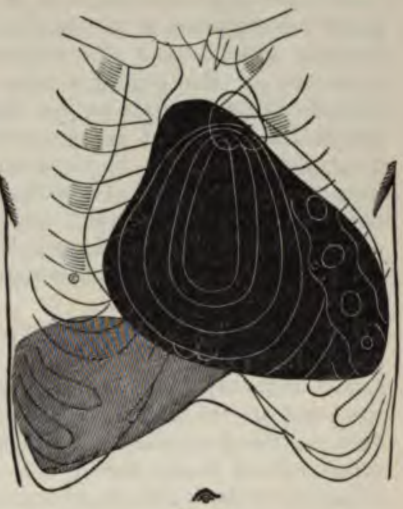

FIG. 41.

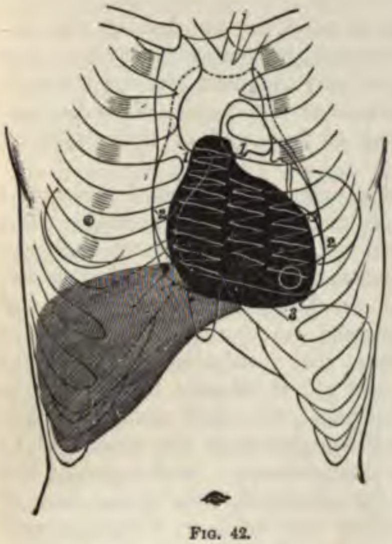

FIG. 42.

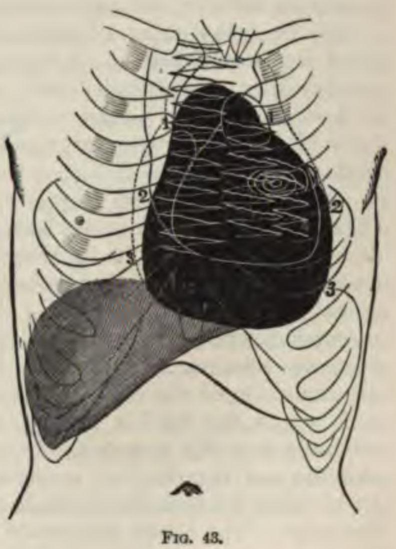

FIG. 43.

of a large quantity of fluid pressing down the diaphragm; or it may be associated with a more median and vertical position of the heart, the aortic arch becoming slightly straightened (Ewart).

Over the pulmonary artery at the base a double beat is sometimes felt, the second being the diastolic shock due to the closure of its valves.

(*b*) The next change to be noticed in pericardial effusion is a progressive weakening of the cardiac impulse from below upwards. This depends mainly upon the amount of the effusion, but partly upon feebleness of the heart's action. When the fluid is in moderate quantity there is often, as just stated, a strong impulse over the upper spaces, its lower and outer boundaries being also well defined. As it accumulates, however, in increasing abundance, and separates the heart from the chest wall, the cardiac movements become more and more obscured, until finally they may be wholly lost, and not perceptible over any portion of the precordial region. This sign is occasionally very striking in a case of inflammatory pericardial effusion when it first comes under observation.

(*c*) It is a disputed question whether pericardial effusion can produce any definite change in the character of the cardiac movements, tactile or visible. Certainly the impulse observed over the upper part of the chest may be more or less undulatory; and a wave-like motion has been described, which can be seen but not felt, and is supposed to be communicated to the fluid by the action of the heart. I must say that I have never been able to recognise this phenomenon positively. Some authorities regard an undulatory impulse as a sign, not in favour of pericardial effusion, but against it.

(*d*) In some cases of pericardial effusion the rhythm of the impulse has been described as lagging behind the ventricular systole in a peculiar way. Irregularity, with or without inequality in the strength of the beats, may become very marked as the result of embarrassment of the heart by a large collection of fluid, and of changes in its walls.

3. One of the most frequent and characteristic signs of pericardial effusion of any extent is an increase in the area of the normal cardiac dulness, with change in its shape and outline; and not uncommonly these alterations are so pronounced as to attract immediate attention in cases of acute pericarditis. The exact quantity recognisable by percussion cannot be definitely stated, and no doubt it varies under different circumstances; but I believe that methodical and careful determination of the cardiac dulness may afford valuable information in cases where the fluid is present in comparatively small quantity. It is necessary to study systematically and thoroughly both the superficial or absolute and the deep or relative cardiac dulness. As the patient lies on his back the increase of dulness is first observed towards the base of the heart. The limits ultimately reached vary much in different cases. The extension takes place chiefly in a lateral and upward direction, the length and breadth of the dulness being thus increased; the former usually preponderating. In most instances it reaches the third cartilage or space, but may extend as high as the second cartilage or first space,

or even above the clavicle. Dr. Sansom maintains that whenever marked dulness extends above the third rib there is a strong probability of pericardial effusion. Over the sternum, which is absolutely dull, as the fluid increases the dulness reaches a higher level than over the costal cartilages, and in extreme cases it may reach its upper margin. From side to side at its greatest width the dulness may extend from an inch or more to the right of the lower part of the sternum, or the right mammary line, to an inch outside the left nipple, or even to the left axilla. In a downward direction it seldom passes below the sixth rib, but in extreme cases it may be made out as low as the seventh or eighth rib, and be indistinguishable from the hepatic dulness. A sign suggested by Dr. Rotch, as valuable in the early diagnosis of effusion into the pericardium, is the presence of dulness in the fifth right intercartilaginous space, due to the accumulation of the fluid in the right corner of the sac; but this sign is by no means invariably to be trusted.

A notable feature of the dulness in cases of considerable pericardial effusion is its shape, which corresponds with that of the sac itself. Thus it narrows from below upwards, assuming a more or less triangular, pyramidal, or, more strictly speaking, pyriform or pear-shaped outline, with its truncated or "peaked" apex above, and its base below, at the level of the lowermost limit of the fluid. The left border has been described as usually somewhat curved, or indented at its upper part, while the right is more nearly vertical. Dr. Ewart well describes the outline of a large effusion as "that of a bag of fluid spreading out at the base." In its diagnosis from cardiac dilatation he lays stress upon the projection of lower angle of the dulness to the right, as well as to the left; a prominent angular outline being made out by careful percussion instead of the normal outline of the heart. When the pericardium becomes extremely distended, the characteristic shape is more or less modified, and may ultimately be altogether lost. Shattuck renounces all faith in the doctrine of a pyriform or pyramidal area of dulness in pericardial effusion, but I differ from him entirely on this point.

In cases of pronounced pericardial effusion the extreme degree of the dulness is very striking. Dr. Sansom insists on the importance of the well-defined transition from the resonance of the lung to such dulness as a factor in the diagnosis of this condition, and in many cases the contrast is certainly very remarkable. In other instances, however, it must not be forgotten that the distended pericardium is overlapped by the margins of the lungs, which yield a superficial resonance; and that its full extent can then be made out only by very careful percussion beyond the limits of absolute dulness. A large effusion imparts an increased sense of resistance to the fingers.

Another important point is that the dulness of extensive pericardial effusion can be made out distinctly towards the left, considerably beyond the position of the apex-beat, which is then only to be recognised by auscultation.

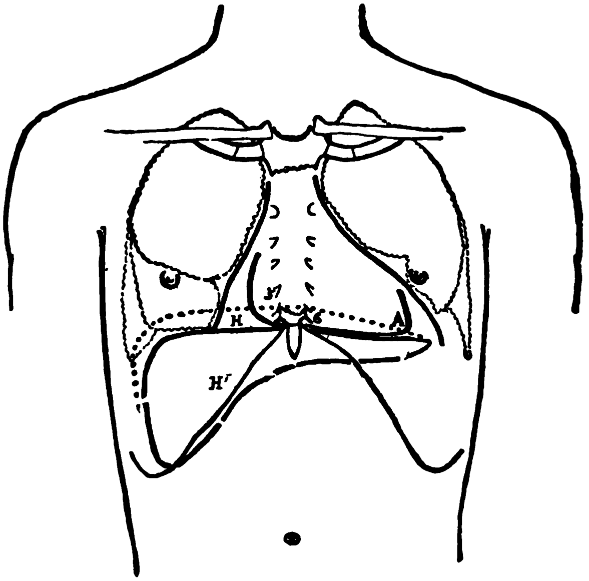

FIG. 44.—Illustrating "Rotch's sign" (dulness in the right 5th space, 5 to H); also contrasting the angles (on either side of H) of the dulness as due respectively to effusion and to dilatation. The heart's outline is normal in size and position. The outer lines are those of the dulness in moderate effusions. The "supra-hepatic line" (dotted) and the "hepatic line" limit the normal "modified" dulness of the liver; and H is placed on the absolute dulness. (After Ewart.)

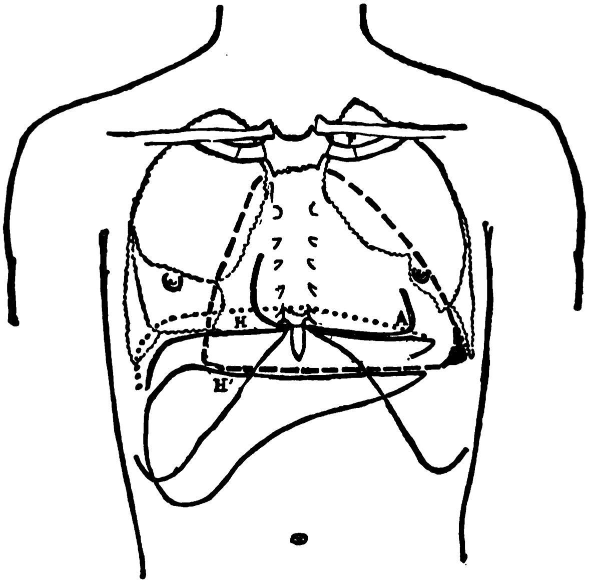

FIG. 45.—Outline of a large effusion, which the pulmonary fringes overlap, and of its total area of dulness. The liver is depressed from its normal level H (infrasternal notch) to the tip of the xiphoid. F shows the position of the finger for the "first rib sign." (After Ewart.)

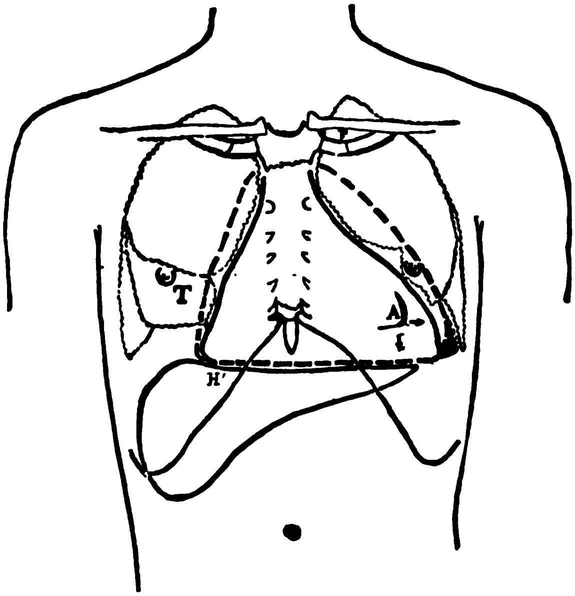

FIG. 46.—Outlines of the total and of the absolute areas of pericardial dulness. A, position of the cardiac apex (5th space) in the effusion. The dulness is shown by the arrows to extend far beyond and below A. The right auricle (not shown) descends with the diaphragm. T, the infra-mammary patch of tubular breathing. (After Ewart.)

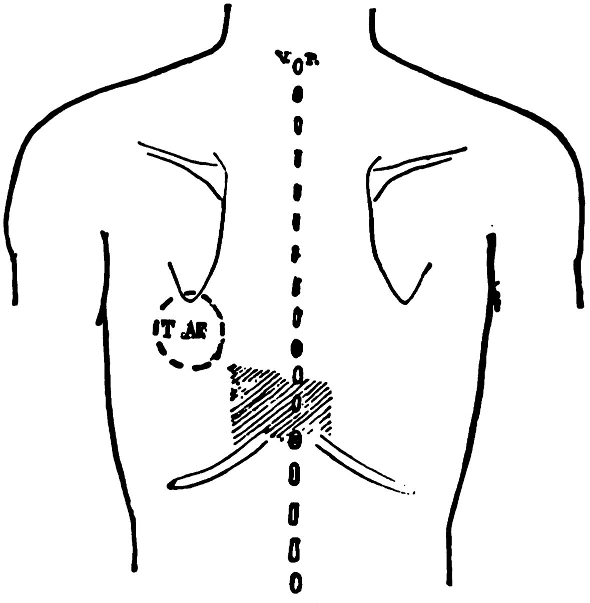

FIG. 47.—The "posterior pericardial patch of dulness" (shaded); and the "posterior pericardial patch of tubular breathing and ægophony." (After Ewart.)

The rapid development of increased precordial dulness while a patient is under observation is strongly in favour of accumulation of fluid in the pericardium, and under circumstances where acute pericarditis might be anticipated this sign must be specially looked for. It may soon become quite pathognomonic, but the possibility of the occurrence of acute dilatation must not be overlooked.

4. The auscultatory signs which may directly result from effusion into the pericardium demand brief notice. The tendency of the fluid itself, as it increases in amount and rises higher and higher, is to weaken the heart-sounds in a progressive manner from apex to base; or they may seem deep and distant. These effects may be due both to imperfect transmission of the sounds through the intervening fluid, and to embarrassment with enfeeblement of the cardiac action. Most commonly in pronounced pericardial effusion the sounds are weak or perhaps inaudible over the region of the normal apex-beat, and for some distance upwards, but become gradually more perceptible towards the base of the heart, where they may be well heard; over the pulmonary artery the second sound may actually be intensified. In cases of extreme effusion the sounds may be practically absent over the whole precordial region.

Some observers have described a basic systolic murmur as a sign of pericardial effusion, the result of pressure by the fluid upon the great arteries. I have never met with such a murmur within my own experience, but it may possibly occur. On the other hand, pericardial effusion may certainly obscure or render inaudible endocardial murmurs previously heard.

5. *Signs connected with neighbouring structures.*—The effects produced on the lungs, especially the left, by a large pericardial effusion, are likely to be indicated by more or less pronounced signs, which, however, will vary in different cases according to their exact nature and degree. The respiratory movements over the upper part of the chest are often obviously excessive, but especially on the right side; and should the fluid be very abundant, a striking contrast will probably be observed between the activity of the two sides, the movements on the left being very deficient. Over the region of absolute cardiac dulness there will be entire absence of breath-sounds, as well as of vocal fremitus and resonance. Beyond its limits there may be hyper-resonance and puerile breathing; and towards the left side the percussion sound is occasionally somewhat tubular, and the breathing bronchial or tubular, with increased vocal fremitus and resonance. Ewart (19) calls attention to a sign which, although not constant, should, he says, be looked for in severe cases; namely, tubular breathing below the right mamma. He describes it as situated usually in the nipple line, a little above the hepatic line, and he states that it is sometimes restricted to expiration. Dry rhonchi of various kinds may be audible in severe and protracted cases, the result of catarrh of the bronchial tubes.

In considerable pericardial effusion the condition of the left lung may give rise to a definite group of signs at the back of the chest on that side; namely, a limited area of deficient resonance or actual dulness, about the size of a crown piece, generally referred to the vicinity of the angle of the scapula, with increased vocal fremitus, bronchial or tubular breathing, and bronchophony or ægophony. Sansom regards these as valuable signs in children and young subjects. Ewart attaches special diagnostic importance to the dulness, which he describes as follows:—"Whenever fluid is effused into the pericardium the normal resonance is modified at the left posterior base in a most definite way. A patch of marked dulness is found at the left inner base, extending from the spine for varying distances outwards, usually not quite so far as the scapular (angle) line, and ceasing abruptly with a vertical outer boundary. Above, its extension is also variable according to the size of the effusion; commonly it does not extend higher than the level of the ninth or tenth rib, and here again its horizontal boundary is abrupt. Its shape is that of a square, and it is quite unlike that of any dulness arising from pleuritic effusion." He attributes this patch of dulness to the altered dorsal relation of the liver, and states further that partial dulness also extends for a short distance to the right of the corresponding vertebræ, and that, when the effusion is considerable, the extension of the patch in the right chest may become almost absolutely dull. With regard to auscultation signs, this writer affirms that over the dull patch to the left of the spine respiratory sounds are found to be absent and the voice sounds feeblé. He locates tubular breathing and ægophony to a patch about two inches in diameter immediately below or slightly to the left of the tip of the left scapula. He concludes that this sign, although not so important as that of the patch of dulness, is very commonly, if not always, present in cases of considerable effusion, and gives valuable confirmation to other signs. The editor of this work tells me that he once found these signs very definitely in a case of a large collection of blood slowly effused into the pericardium from a ruptured coronary artery. The patient, a lady of some threescore, lived about sixteen hours from the onset of the symptoms.

As previously stated, pleural effusion on one or both sides is not uncommon as a consequence of a large collection of fluid in the pericardium; in which case the signs will be modified accordingly. When it begins on the right side the contrast may be helpful in diagnosis. Signs indicative of downward displacement of the liver are very pronounced in cases of extensive pericardial effusion, and there may also be some degree of enlargement due to venous congestion.

6. *Effects of change of posture.*—The study of the effects produced by changes of posture upon the chief signs just discussed has generally been regarded as important in the diagnosis of pericardial effusion. In a large proportion of cases these signs are so definite that it is quite unnecessary to test them in this way, and under such circumstances it is highly dangerous to place the patient in the sitting or erect posture; such disturbance may even prove immediately fatal.

The following are the chief modifications in the signs produced by changes of posture, which are regarded as of more or less diagnostic value. It may happen that the impulse is not perceptible in the recumbent position, but becomes evident when the patient is made to sit up or bend forwards. Increased mobility of the apex-beat with change of posture has also been looked upon as important, but certainly this is very untrustworthy, to say the least. The effects of position upon the dulness have been more particularly insisted upon as evidence of pericardial effusion, and in doubtful cases may be worth studying. It is increased in extent, especially at its upper part, in the sitting posture, and still more if the body is bent forwards. It may also be modified in a lateral direction, as the patient turns to either side. The relative loudness of the cardiac sounds or of endocardial murmurs might also possibly be similarly influenced. Modifications of the signs observed in connection with the left lung posteriorly are also said to be produced by change of posture. Sansom writes: "If the patient bend well forward, or assume the knee-elbow position for a short time, the dulness disappears, and in its place a tympanitic sound is elicited; in like manner the former auscultatory signs of lung-consolidation vanish, or only slight crepitant rales are heard, which also very soon become inaudible. The former signs return when the vertical position of the body is resumed."

It must again be insisted upon that the physical signs of pericardial effusion vary much in different cases, and most of the special signs described by Ewart and others apply rather to cases in which it is so abundant as to raise the question of operative interference, when a positive diagnosis is obviously of extreme importance. With some of them I am not familiar, and I hardly think that any of them are absolutely trustworthy. Shattuck denies the existence of the dorsal signs of pericardial effusion. It may be mentioned that skiagraphy has been employed for the diagnosis of pericardial effusion and other conditions of this sac, but it is too early at present to attempt any definite statements as to its real and positive value.

Stage of absorption.—During the progress of absorption of inflammatory pericardial effusion the signs indicative of this condition progressively diminish, until the phenomena become practically normal, or point to the formation of adhesions. The friction signs, if they have been obscured by the effusion, return for a while; or they alter in their situation, intensity, extent, and characters. Friction-sound in most cases increases in a downward direction as the fluid declines (Sibson). It lasts a variable time. Friction-fremitus may at this period be noticed for the first time; and the sound is often rough and creaking or churning. The dulness diminishes more or less rapidly from above and laterally; while at the same time the sounds become more distinct. It must be remembered that one or more relapses may take place, with further increase of the fluid, the signs of which then return, again to subside as the fresh effusion becomes absorbed. What the ultimate position of the heart and the apex-beat will be depends on the course of events. As a rule, in simple and uncomplicated cases of pericarditis it returns to its normal situation, but this return may be prevented by adhesions, by the effects of endocarditis, or by other causes. The signs indicative of adherent pericardium will be separately considered, but it may be remarked that in not a few instances, if carefully watched for, they can be traced in process of development during the period of convalescence.

Course and terminations.—As already stated, acute pericarditis presents much diversity in its clinical history, and it does not follow any uniform course. When, however, the symptoms and physical signs discussed in the preceding pages have been adequately and intelligently mastered, they can be studied with advantage in individual cases on the lines indicated. Among the chief circumstances which influence the nature, severity, and combinations of the symptoms, may be mentioned the causation of the pericardial inflammation, and the character of the general disease to which it is secondary; its intensity and rapidity of progress; the characters and amount of the inflammatory products, especially of the effusion; the presence of previous organic changes affecting the heart or pericardium, or of other chronic intrathoracic diseases; and the association of the pericarditis with endocarditis or myocarditis, or with pleurisy or pneumonia.

Attempts have been made by Stokes and others to classify cases of acute pericarditis into groups, according to the intensity of the symptoms, and the morbid changes affecting the pericardium and heart associated therewith; but distinctions of this kind are quite arbitrary, and have no practical foundation or value. It may be affirmed that as a rule the clinical phenomena are not so pronounced or so grave as is commonly supposed, or as the older writers used to describe. Not uncommonly the symptoms are not at any time prominent; they may be practically latent, or they may quickly attain some degree of severity, and as speedily subside. Shattuck, indeed, specifies "latency" as the most characteristic clinical phenomenon of pericarditis. In some instances one or more relapses occur, with corresponding increase of the symptoms after their subsidence. Acute pericarditis may run a favourable course in a few days, even when there is considerable effusion, which then undergoes rapid absorption. The entire duration of the majority of cases is from eight or ten days to a fortnight, but not uncommonly longer; convalescence may not be established for three to six weeks or more, or the disease, after beginning more or less acutely, may afterwards assume a subacute or chronic course. As a rule it terminates in recovery, so far as the immediate result is concerned, and no doubt in a considerable proportion of cases the restoration is practically complete; but in not a few instances definite organic changes are left behind, the effects of which are sooner or later revealed, it may be within a short period. Sometimes the patient can hardly be said to recover, a condition of obvious chronic pericarditis being established, with well-marked symptoms and physical signs which will be considered later. It is impossible to make any definite statement as to the direct fatality of acute pericarditis, and the more important points bearing upon this matter will be more conveniently referred to under prognosis. It may be affirmed, however, that death is seldom due solely to this affection, though evidences of pericardial inflammation may not uncommonly be found at post-mortem examinations, or it may partly contribute to the fatal result. Occasionally acute pericarditis assumes a very grave aspect from the first, advancing with great rapidity, exhibiting extremely severe symptoms, and ending in death within a short time, it may be even in less than twenty-four hours; but such a course of events only occurs under special circumstances, and mainly in hæmorrhagic cases.

The course of rheumatic pericarditis in children is described by Dr. Cheadle as usually subacute, chronic, recurrent. It frequently merges into the condition of pericardial adhesion and its consequences, with their attendant phenomena. [For fuller details the reader is referred to the article, "The Acute Rheumatism of Childhood," vol. iii. p. 44 *et seq.*]

Diagnosis.—Several important matters bearing upon the diagnosis of acute pericarditis have been sufficiently dealt with under its clinical history, especially in the discussion of its physical signs; and in further consideration of this part of the subject, I propose merely to draw attention to its more prominent and important aspects.

An ordinary case of acute pericarditis arising in the course of definite rheumatic fever ought to present little or no difficulty in diagnosis, if due attention be paid to the symptoms and physical signs. Remembering, however, that the inflammation may supervene very insidiously in this complaint, and when the joint-symptoms are not pronounced, it is necessary, whenever any rheumatic condition is suspected, to be constantly on the watch for its appearance. Nor must we forget that pericarditis may be the first indication of such a condition. From these points of view it is a disease to be particularly watched for in children, though in such subjects its symptoms and signs, as well as its mode of progress, may be very anomalous, even where there is well-marked or perhaps a large pericardial effusion, a state of things, however, which ought not to occasion any difficulty to an intelligent and practised clinical observer. The occurrence of acute pericarditis in other than rheumatic cases may easily be overlooked by an incautious observer, but it should be thought of at any rate as a possible complication of Bright's disease, or of pneumonia or pleurisy.

Assuming that the diagnosis of pericarditis has been made, it is obviously very important to determine, within due limits, and without endangering or needlessly distressing the patient, the actual morbid conditions present, and more especially the amount and characters of the fluid effusion, as well as the changes which take place during the progress of the case. Most of these points can be positively made out by physical examination only, conducted on the lines already explained. It must not be forgotten that extensive friction-sound is not incompatible with a very abundant effusion. The rapid extension of general pericardial adhesion in some cases is also worthy of note, especially in children. The probability of the fluid being hæmorrhagic, suppurative, or ichorous is mainly founded on the conditions with which the pericarditis is associated, and on the general symptoms; yet these may be in no way characteristic.

What other conditions of the pericardium, or of the heart itself, are apt to be confounded with pericarditis? A dropsical accumulation—*hydropericardium*—may certainly be mistaken for an inflammatory effusion, especially if it be abundant. However, the circumstances under which it occurs, the fact that it usually follows hydrothorax, the absence of symptoms of pericarditis and of any friction phenomena, and, as a rule, the comparatively small amount of the effusion, will usually enable a diagnosis to be arrived at readily. A morbid growth involving the pericardium has more than once been mistaken for pericarditis with effusion. The distinction of pericarditis from endocarditis at an early stage is mainly founded on the differences between the tactile and auscultatory signs already discussed, but the symptoms may also help. When marked effusion occurs, any previous difficulty is cleared up. Of course the frequency with which the two diseases are associated together, especially in children and young subjects, must always be borne in mind. Implication of the heart substance is indicated by evidences of serious embarrassment and feebleness of its action, and when grave symptoms arise in the course of pericarditis, changes in the muscular tissue of the heart may be regarded as highly probable. Much has been written about the difficulties of distinguishing between pericardial effusion and cardiac enlargements, especially dilatation, but in my opinion they have been greatly exaggerated, when we remember that due consideration is to be given to all the facts of an individual case. It is possible, indeed, that a much dilated heart, especially if associated with extensive adhesions, might be mistaken for effusion; and such a mistake has actually happened several times, the heart having been punctured in an operation for the removal of a supposed pericardial collection of fluid. Difficulty might also arise when acute dilatation with rapid adhesion occurs in pericarditis, instead of effusion. Should inflammatory effusion supervene where the heart is enlarged, and the pericardial sac distended, the diagnosis might likewise be obscure; as well as when acute inflammation involves a narrow area of the pericardium, the rest of the sac being obliterated by previous adhesions.

The diagnosis of acute pericarditis from neighbouring conditions is, as a rule, quite easy. Occasionally the distinction between this complaint and pleurisy might be difficult, and certainly this applies to the friction-sound. A superficial exo-pericardial sound, or even a fremitus produced in the mediastinal cellular tissue, might also simulate pericardial phenomena. The only circumstance in which a pleural effusion is at all likely to resemble one in the pericardium is when it happens to be peculiarly limited by previous adhesions. It has been stated that such conditions as pneumonia, phthisis, aneurysm, accumulation of fat, or intrathoracic tumour might be mistaken for acute pericarditis, but I have certainly never met with any difficulty of this kind. It must not be forgotten that this disease may be associated with other inflammatory affections within the chest, or be secondary to certain adjacent morbid conditions.

Prognosis.—Acute pericarditis must be regarded as a serious disease, though in uncomplicated cases the immediate prognosis is usually favourable. The mortality is comparatively small, but it is not practicable to give any definite percentage of deaths. Much depends upon the conditions with which the disease is associated, rheumatic cases being seldom immediately fatal. It is far more dangerous when it supervenes in connection with Bright's disease or other such grave chronic maladies, and is then likely to end fatally. Septic cases of all kinds are also very grave. Seeing that pericarditis and endocarditis so often go together, the prognosis under such circumstances must be guided by a due consideration of the effects of the combination in each particular case; but obviously it must always be more serious, especially if the myocardium is involved also. When there are other acute inflammatory affections within the chest, in addition to those implicating the heart and its covering, the danger is very imminent.

Among the factors influencing the immediate prognosis in individual cases the following are worthy of note:—Pericarditis is very serious in infants and young children; and the very fatal form described by Sturges, attended with grave nervous symptoms, and ending in rapid adhesion, must again be specially mentioned. In old people also the danger is decidedly greater. Previously impaired health, or a weak condition of the patient, and particularly the presence of old heart trouble or other chronic diseases, especially intrathoracic, may further complicate matters. The character and amount of the morbid products in acute pericarditis greatly affect the prognosis. The danger is obviously more serious in proportion to the quantity of fluid effusion; as well as if there be reason to believe this to be of a hæmorrhagic, purulent, or ichorous nature. Due observation and study of the symptoms may afford important indications. Among those of more or less grave import are serious dyspnœa, especially if amounting to orthopnœa, with signs of cyanosis or asphyxia; greatly embarrassed or very feeble or irregular cardiac action, with corresponding pulse, and tendency to faintness or syncope; hyperpyrexia; dysphagia; severe vomiting; marked prostration; and pronounced cerebral or other nervous disturbances. The general appearance and the expression of the face and eyes are often useful guides to the immediate prognosis. It must never be forgotten that sudden death from syncope may happen in cases of large effusion into the pericardium, especially if the patient is made to sit up, or to change his posture for the purpose of physical examination. Finally, the mode of treatment materially influences the immediate prognosis in acute pericarditis. Undue activity may certainly do much mischief; but, on the other hand, a dread of energetic measures, when circumstances demand them, may as certainly lead to a fatal result.

The remote prognosis in a case of acute pericarditis always demands special attention, though it is often impossible to give a positive opinion on this point until the course of events has been watched for some time. I believe that the general tendency is to take too favourable a view of the ultimate prognosis, and not adequately to recognise the importance of the after-effects of the inflammatory changes. Such after-effects are met with in a considerable number of cases, and may be very serious, as will be pointed out in relation to pericardial adhesions. They are more likely to give trouble in proportion to the amount of lymph effused; to its presence over the exterior as well as the interior of the pericardium; to the slow or subacute progress of the disease; and to the formation of pus.

Treatment.—The treatment of each individual case of acute pericarditis demands careful and intelligent consideration, and it is decidedly a mistake to follow any regular routine plan, or to adopt needlessly active measures. When it occurs in connection with rheumatism it may not be requisite or desirable to change the previous treatment in any way, but much will depend upon the nature and degree of the morbid changes which the pericardial inflammation produces. The administration of salicylates is not contra-indicated, and many believe that they help in averting the complaint, but certainly their use requires caution. Dr. Gee has recently stated that large pericardial effusions are much less commonly met with now than formerly, and suggests that this may be due to the use of salicylates. Whether it be possible to prevent the development of pericarditis in rheumatic cases is a doubtful question, but at any rate complete rest, avoidance of chill, and due protection of the precordial region may help in this direction. Should there be a tendency to much cardiac excitement, I believe it is a good plan to administer opium or morphine as a preventive measure in suitable cases, the effects being of course duly watched.

When acute pericarditis has actually arisen, the treatment must be guided by circumstances. In every case the patient must be kept as much as possible at rest, and must not be unduly disturbed or moved for the purpose of physical examination. Posture must be intelligently studied in relation to the pericardial conditions, the symptoms, and the feelings of the patient. As fluid accumulates it is often necessary to have the head and shoulders raised; but, if so, the patient should be propped up comfortably and effectually supported; this arrangement requires special attention. The judicious administration of nourishment constitutes an important part of the treatment in many instances; and alcoholic stimulants, especially brandy and champagne, are often needed; the quantity must be determined by the requirements of each individual case, as judged chiefly by the degree of general weakness or depression, and the cardiac action and pulse. In bad cases a considerable amount may be required.

The treatment of acute pericarditis in the early stage has for its objects the relief of pain and restlessness, the calming of the heart's action, and the arrest or control of the inflammatory process. The practice of bleeding and giving calomel, formerly adopted by many as a matter of routine, need only be mentioned to be absolutely condemned; nor in my opinion can anything favourable be said for the use of cardiac depressants, such as antimony, aconite, or green hellebore. In suitable cases advantage may certainly be derived sometimes from the application of a few leeches. As a rule, however, efficient poulticing over the front of the chest gives most relief at first, and answers best in the majority of cases, cotton-wool being afterwards applied. Fomentations or spongiopiline are also convenient applications. I have thought that the application of a blister over this region at an early period has in a few instances checked the progress of the inflammation, but it is easy to be deceived in this matter. The application of cold, by means of ice-bags over the precordium, is strongly advocated by Dr. Lees and others, but this treatment certainly requires caution. Should the pain be severe, opium may be given, Dover's powder being a useful preparation; or morphine may be administered subcutaneously, and repeated as occasion demands. There is no harm in judiciously applying anodynes, such as belladonna, over the precordial region; but I doubt whether they are really beneficial.

The treatment of pericardial effusion must be guided by its quantity and mode of progress. If it is not abundant, and shows the natural tendency to become absorbed quickly, no special measures are needed. Otherwise it may be desirable to apply a blister, or even two or more in succession. Some prefer applications of tincture or liniment of iodine as counter-irritants; others advocate the inunction of mercurial ointment or oleate of mercury. The internal administration of iodide of potassium or sodium may be of service, combined with tincture of digitalis. Iron preparations may also be helpful, especially the tincture of perchloride; and a combination of tartrate of iron with the iodide has been recommended. Very active measures to promote absorption are certainly to be deprecated; and, when the effusion is large, special care must be taken not to make the patient sit up suddenly lest fatal syncope should occur.

In all cases of acute pericarditis it is necessary to watch carefully the action of the heart and the pulse from the point of view of treatment. I have already expressed my opinion that at no time is it desirable to give cardiac depressants. Some authorities recommend the administration of tincture of digitalis from the outset, but I do not think that a routine use even of this drug is desirable. However, should there be any indication of cardiac weakness, or a marked want of tone in the arteries, with dicrotism of the pulse, the tincture should be given every three or four hours in ten-minim doses, its effects being duly watched. Strychnine affords valuable help in bad cases, and may be combined with digitalis; or it may even be thought desirable to employ subcutaneous injections of strychnine and digitalin. Of the use of strophanthus or other cardiac tonics in pericarditis I have no experience. As temporary stimulants, ammonia and ether might be of decided service in some cases; or possibly subcutaneous injection of ether. Of course alcoholic stimulants are often of the greatest assistance, and large quantities of champagne or brandy may be demanded. The administration of the agents mentioned in the preceding remarks needs the most careful supervision, and they must not be employed indiscriminately or rashly, for it may be desirable at any time to diminish the dose, or to stop them. Special care must be taken in the treatment of children.

Pericarditis not of rheumatic origin must always be treated as a part of the general condition with which it may be associated, such as septicæmia, tuberculosis, or renal disease; and in its association with endocarditis, or with other intrathoracic inflammatory affections, the knowledge, experience, and judgment of the practitioner will often be severely taxed, though not uncommonly but little can be done. Much difficulty may also be experienced in the treatment of symptoms, which must be conducted on ordinary principles, though considerable discretion and caution are demanded in carrying them out. Among the most important symptoms which may need attention are dyspnœa, especially if accompanied with a tendency to cyanosis or apnœa, dysphagia, severe vomiting, restlessness and sleeplessness, delirium or other cerebral disturbances, and high fever. Dr. George Balfour recommends chloral hydrate as a sedative and antiphlogistic along with digitalis; it is, however, a depressant of the heart, and must at any rate be cautiously used. Want of sleep is a very trying symptom, but such remedies as sulphonal, trional, or paraldehyde in suitable cases may help us better. Subcutaneous injection of morphine may be imperatively demanded, even if risky. Dr. Cheadle speaks highly of nepenthe for children. Inhalation of oxygen may help the breathing in some cases. The measures to be adopted to bring down temperature, especially hyperpyrexia, must be determined by circumstances. Difficulty in swallowing may, perhaps, be relieved by making the patient bend forwards, so as to relieve the œsophagus from the pressure of the distended pericardium; but special care must be exercised in doing this. The bowels need due regulation; and in bad cases it is important to see that the bladder is properly emptied.

The quantity of a serous effusion, and the imminent danger to life resulting therefrom in exceptional cases, may raise the question of surgical interference, but I cannot agree with those who are too ready to resort to paracentesis for pericardial effusion. It is rarely required at any rate in rheumatic pericarditis. Dr. Clifford Allbutt (1) was the first to introduce as a practice the operation of paracentesis pericardii into this country in 1866, when it was successfully performed on a patient of his by Mr. Wheelhouse; the patient, who was moribund at the time of the operation, made a good recovery. In another case it was performed for him by Mr. Teale in 1869. For a full description of the operation reference must be made to surgical works (see especially *Surgery of the Chest*, by Mr. Stephen Paget), and it will only be necessary to refer here to two or three practical points. To determine that fluid is really present an exploratory puncture may be made, in the first instance, with a hypodermic syringe; and, as a dilated heart has even within a recent period been actually perforated for a supposed pericardial effusion, this precaution is certainly advisable in any obscure case. Some prefer even to make an incision down to the pericardium. The fluid is best removed by means of an aspirator with antiseptic precautions, but the instrument must not be too powerful, as the effusion needs to be taken away very gradually. Some operators prefer a small trochar and canula. Either the fourth or fifth left interspace is usually selected, at a distance of an inch (Dieulafoy) to 2 or 2½ inches from the margin of the sternum; but the exact spot may vary with circumstances. The puncture has even been made on the right of the sternum. Rotch recommended the fifth right interspace. The late Marcus Beck recommended the use of a No. 2 needle, which he passed obliquely upwards and inwards, taking care to turn on the vacuum as soon as the eye is covered. The moment the fluid gets into the syringe the needle must be held steadily until the flow ceases. The patient must be in the recumbent posture during the operation, and its effects carefully watched. When pericardial is associated with pleural effusion, the removal of the latter may sufficiently relieve all urgent symptoms, but if it tend to return it may then become necessary to relieve the pericardium also. The subject of paracentesis pericardii has been very ably dealt with in a paper by Dr. Samuel West, who gives a tabular summary of eighty cases thus treated up to 1883. Subsequently it has been discussed by Sir T. Grainger Stewart and others, and many scattered cases have been recorded.

The management of cases of pericarditis during convalescence is a matter requiring due consideration, especially in relation to the formation of adhesions. Personally I have been disposed as a rule to enforce prolonged rest, but some years ago Dr. Cantlie drew attention to the desirability of encouraging exercise after an attack of acute pericarditis in young subjects, with the view of exciting the cardiac action, and thus helping to make the adhesions loose and filamentous. This question has usually to be considered in relation to the presence or absence of endocarditis and its consequences, as well as the state of the cardiac walls; so no general rule can be laid down, and every case must be studied on its own merits.

II. SUPPURATIVE PERICARDITIS; PYOPERICARDIUM

The formation of pus within the pericardium has already been mentioned under acute pericarditis, but it will be expedient briefly to consider this condition separately, including also those cases in which the fluid is of an ichorous kind.

Etiology and Pathology.—Pyopericardium is occasionally acute in its manifestation, but is much more commonly the result of a subacute or chronic process. It is very rarely the outcome of an ordinary acute pericarditis, either primary or rheumatic, being then a late or secondary phenomenon, a serous or sero-fibrinous effusion gradually changing into a more or less purulent collection. In the large majority of cases, however, the circumstances under which such a collection is met with are peculiar, and it may not only be formed within the pericardium, but in some instances is partly due to the bursting of a neighbouring accumulation of pus into the sac. Pathologically it is associated, of course, with pyogenetic organisms. It has been stated that the production of pus within the pericardium is favoured by abundant exudation, and the extensive formation of new blood-vessels in its substance. The longer a pericardial effusion remains unabsorbed the more likely it is to become purulent.

Pyopericardium occurs most frequently in cases of pyæmia or septicæmia of all kinds; thus it may appear as a complication of certain of the eruptive fevers. It has been said to be associated particularly with injuries and diseases of bones, such as osteomyelitis and acute necrosis. Purulent pericarditis is more likely to occur if an abscess has previously formed in the myocardium, but this is by no means necessary. Very rarely it appears to have been secondary to malignant endocarditis. In another class of cases pyopericardium is due to the rupture of a neighbouring collection of pus into the sac, especially of an empyema; or it may even be set up by contaminated air, which has entered through a perforation. Exceptionally it results from the extension of empyema, low forms of pleuro-pneumonia, neighbouring ulcerative or gangrenous diseases or abscesses, or possibly peritonitis. The pericarditis associated with Bright's disease is believed to have a special tendency to the formation of pus; and a similar tendency has been attributed to the tuberculous variety. Among the cases of operation collected by Dr. Samuel West (53), however, in no instance of tuberculous pericarditis was the effusion purulent. Such a condition may be associated with pulmonary phthisis, owing to the rupture of a cavity into the sac. Pyopericardium is far more common in young subjects, and in males.

Anatomical characters.—As the name indicates, the essential change in pyopericardium is the presence of pus in the sac. It may be in small amount, or the accumulation may be very large; in the latter case it will produce the same mechanical effects upon the heart and neighbouring structures as other forms of effusion. In Dr. Dickinson's most interesting case (17) as much as 19½ oz. were drawn off at one time; and in Dr. Samuel West's case 14 oz. and 16 oz. were successively removed. It may collect entirely in the posterior portion of the pericardium, the anterior surfaces being adherent as in a case of Sears. The pus is usually laudable and inodorous, but may often be shreddy, flocculent, curdy, or even membranous; and it may be mixed with lymph. Exceptionally and under particular circumstances it is offensive, and may be of an "ichorous" nature, very foul or even stinking. It may also become fœtid after operation. Occasionally there is an admixture of blood. In most cases the surface of the membrane becomes like that of the granulating surface of a wound. Rarely part of the parietal pericardium becomes destroyed, and perforation takes place, which has even ended in a superficial fistula;

but at the present day such a termination could hardly be permitted to occur. There seems to be good reason to believe that a purulent collection in the pericardium may in exceptional instances be absorbed, leaving dense and thick adhesions; or some of it may remain in an inspissated condition as a yellowish white paste, limited and encapsuled by adhesions, consisting of caseous material, in which calcareous particles may afterwards form; thus it may ultimately be converted into a chalky pulp, or even into a hard calcified mass.

Clinical history, Diagnosis, and Prognosis.—Speaking generally, the symptoms and physical signs of pyopericardium will be more or less like those of serous effusion, modified not only by the quantity of the pus, but also by the circumstances under which it has formed. It will only be necessary, therefore, to draw attention to certain special points in the clinical history of this condition. When it supervenes in an ordinary case of acute pericarditis, there are no trustworthy indications of a change from a serous or sero-fibrinous effusion to one of a purulent nature; but if the course of the case happens to be prolonged, such a deterioration would be suggested if fever, perhaps of a septic type, persist. Pyrexia may, however, be entirely absent. Considering the circumstances under which pyopericardium occurs, it is easy to understand how insidiously it may set in; its symptoms, if any, being entirely overshadowed by those of septicæmia: thus it often remains undiscovered until the necropsy, especially if the amount of pus be small. In cases of this kind symptoms of serious interference with the respiratory and circulatory functions may show themselves suddenly; and on examination be found to be due to a large but previously latent purulent collection in the pericardium. General symptoms are of little or no value in the diagnosis of pyopericardium. In some of the most pronounced cases neither rigors, pyrexia, nor sweating have been present. Œdema of the legs seems not to be uncommon, but probably is not more frequent than in connection with other large pericardial effusions and their consequences. It may be noted here that œdema over the precordial region may suggest the purulent nature of such an effusion.

With regard to the *physical signs*, the absence of friction-sound throughout cases of purulent pericarditis has been noted by careful observers; or it may be very indefinite and transient. Whether this sign be usually absent, as has been affirmed, it is difficult to say; at any rate it cannot be relied upon in diagnosis. The ordinary signs indicative of pericardial effusion will be evident on examination, in proportion to the amount of the pus. Should gas be present at the same time, the phenomena associated with this combination will probably be noted, but these will be considered separately.

From the foregoing remarks it will be gathered that the diagnosis of pyopericardium is extremely uncertain, and often impossible. Should there be evidence of effusion into the sac, its purulent nature can only be determined positively by the aid of the exploring needle or other apparatus, by which a specimen can be obtained for examination. Some such instrument should be used at once if there be any reason to suspect the presence of pus.

The prognosis of pyopericardium is necessarily grave, especially on account of the conditions with which it is associated. In suitable cases, however, efficient operative interference gives reasonable hope of recovery; and some remarkable results have been thus achieved by modern surgery.

Treatment.—The treatment of pyopericardium is entirely surgical, and it would be quite beyond the province of this article to attempt to discuss the important questions involved. Suffice it to say that mere paracentesis is of no use; the operative procedures adopted must be thorough and bold, and should be carried out as promptly as possible. Free incision, with drainage and due antiseptic precautions, is the method of treatment usually practised.

III. CHRONIC PERICARDITIS; CHRONIC EFFUSION; PERICARDIAL ADHESIONS AND THICKENING

The cases which come within the category of chronic pericarditis may be arranged for practical purposes under two groups; namely, those of—(1) Chronic effusion; (2) Pericardial adhesions and thickening. These conditions are in exceptional instances more or less combined, but it is needless to make an independent group of such complex cases. It will be sufficient to discuss separately the two main divisions just indicated.

1. *Chronic pericardial effusion.* — This morbid condition requires but brief comment. It occasionally happens that acute or subacute inflammatory effusion into the pericardium remains chronic, though fluctuating in amount; or it may return again and again after paracentesis. In rare instances even a simple pericarditis is chronic from the outset; but this course of events is observed chiefly in elderly persons, and there is reason to believe that in some of these cases the effusion is originally a mere hydropericardium. Chronic pericarditis is more likely to be of a hæmorrhagic or purulent nature; or it may be associated with new growths, especially tubercle or malignant disease. Dr. Samuel West mentions a remarkable case of supposed mediastinal cyst, which was tapped several times during a period of four years; the fluid removed on the first occasion deposited a large amount of cholesterine; on post-mortem examination it proved to be a chronic pericardial effusion. In very exceptional instances an accumulation of this nature originates a diverticulum of the pericardium.

Clinically, chronic pericardial effusion does not, as a rule, give rise to any prominent symptoms; practically it is only recognisable by the physical signs already described. In prolonged cases, owing to the changes produced in the pericardium and the walls of the heart, the circulation becomes more or less seriously obstructed, with the usual symptoms, including dropsy. The *treatment* of this condition must be conducted on the general principles applicable to different kinds of pericardial effusion, some operative procedure being generally required; but each case must be dealt with on its own merits.

2. *Pericardial adhesions and thickening.*—The conditions coming under this head are of much pathological and clinical importance, and are worthy of far more attention than they generally receive. It is a familiar fact that they are frequently met with at necropsies in various degrees, when they have not been diagnosed during life. It may be acknowledged at once that their diagnosis is often, for obvious reasons, impracticable, or may be a matter of great difficulty or mere surmise; not uncommonly, indeed, there is no reason whatever even to suspect their presence. On the other hand, to teach that the diagnosis of adherent pericardium is impossible is absolutely wrong and misleading. If pericardial changes of this nature were always borne in mind and systematically looked for, they would be recognised much more frequently than they have been hitherto; as a matter of fact they are seldom even suspected in the ordinary routine of practice, and are therefore necessarily overlooked. Not uncommonly they can be positively demonstrated by physical examination; while in other cases their presence may be reasonably inferred. Dr. John Broadbent, in his valuable monograph on "Adherent Pericardium," duly recognises this truth, and writes: "The comparative rarity with which the existence of adherent pericardium is diagnosed may be accounted for in many instances by the fact that it is not thought of. Especially is this the case when it is associated with valvular disease, for the valvular lesion is judged to be sufficient to account for the symptoms that arise."

Etiology.—The various conditions of the pericardium now under discussion are always of inflammatory origin, and in the large majority of cases they are the remains of one or more acute or subacute attacks of pericarditis, of which there is often, but not necessarily, a definite history. As was mentioned in relation to this disease, extensive adhesions may rapidly form in the stage of fibrinous exudation, especially in children; and if the termination be not fatal, they become organised and permanent. Most commonly, however, they are formed after the absorption or removal of fluid effusion. As might be anticipated, pericardial adhesions are likely to be more firm and extensive in proportion to the number of attacks of inflammation, and to their duration. After a first attack partial adhesions may form, which in subsequent attacks become extensive or general. When a pericarditis beginning acutely assumes a prolonged and chronic course, they are usually well marked, and again when the effusion becomes purulent. The occurrence of acute inflammation over the external surface of the pericardium leads to the formation of adhesions between this structure and the chest wall, the pleuræ and, sometimes, the posterior mediastinal structures or the spinal column.

An important group of cases in which pericardial adhesions and thickening occur are those which are chronic from the outset, and in these cases they are particularly liable to be overlooked. They may naturally be expected when an inflammatory effusion runs a chronic course through, out; but the cases which must be more especially borne in mind are those in which there has been no such effusion, but the morbid changes leading to the pericardial conditions have taken place slowly and imperceptibly. Some of the "white patches" are of this nature, but the most striking cases are those in which a chronic inflammatory process extends from neighbouring structures, particularly in connection with pleurisy or pulmonary phthisis. Adhesions are also usually associated with new growths involving the pericardium, which are practically either of a tuberculous or malignant nature. When the changes leading to these conditions have once started, it seems highly probable that they may extend and increase considerably, as the result of a continued chronic process, which may be regarded as inflammatory, and leads to a progressive hyperplasia of fibrous tissue. In this way it may possibly happen that an adhesion may, as it were, grow through the parietal portion of the pericardium from within outwards or from without inwards, and thus ultimately fix it more or less extensively on both aspects.

Pericardial adhesions may be met with at all ages. They have been observed in very young infants, and even in new-born children, when they are attributed to pericarditis occurring during fœtal life.

Anatomical characters and effects.—It would not serve any useful purpose to describe in detail the numerous and varied aspects under which pericardial adhesions present themselves, but a comprehensive knowledge of the more important groups of cases in which changes of this kind are met with is of decided practical advantage. Before attempting any such classification it will be well to point out that the adhesions are either partial or general; internal or external, or both; that they differ much in length, toughness, and firmness, and are often accompanied by more or less pericardial thickening, which may reach an extreme degree. In exceptional instances there is much thickening, with little or no adhesion between the surfaces. Structurally the morbid formations now under consideration consist either of cellular or fibrous tissue—*pericardial fibrosis.* Sometimes they are associated with the encapsuled remains of fluid, thickened pus, soft caseous or chalky pulp, or dry brittle calcareous concretions, which may attain a considerable size. As already stated, an adherent pericardium may itself undergo calcification. The effects which the morbid changes may produce upon the heart and vessels must be considered separately.

The groups under which I propose to arrange the cases, as they have come under my personal observation, are as follows:—

(*a*) In a large proportion of instances there are merely partial and small adhesions between the contiguous surfaces of the pericardium, it may be in different portions of the sac at the same time. Usually such adhesions assume the form of filaments or threads, or of bands, often of considerable length, stretching between the two surfaces. They may be delicate and cellular, or firm and fibrous, sometimes attaining the thickness of a finger or more. Occasionally adhesions occur in circumscribed closely adherent spots or patches. Ultimately the bands often give way by stretching and attenuation, their remains hanging loosely within the sac, especially near the apex of the heart. The situation, extent, and characters of localised pericardial adhesions are affected by the degree and range of the movements of different parts of the heart and arteries; the relation of the heart to the pericardium; and the effects of gravitation of the organ within the sac in cases of effusion. According to Sibson, they are more frequent a little above and to the left of the apex, and along the line of the ventricular septum; at the outer border of the left ventricle, and the outer side of the right auricle; along the posterior surface of the left auricle and of the ventricles which rest upon the sac; and over the great arteries at their higher part. In several instances he noticed that a patch of the right ventricle, to the right of the septum and midway between the pulmonary artery and the lower border of the ventricle, was adherent, when the rest of the ventricle was free; this being the part of least extensive movement.

(*b*) A second group of cases may be made to include those in which an extensive or general internal adhesion exists between the pericardial surfaces, the external surface being quite free; and this group may be subdivided into cases without and with thickening. Here again many varieties are observed in individual instances, and in the same case the adhesions often differ in their characters over different parts of the pericardium. They may be in the form of fibrous threads or bands, more or less loose and long, and interfering but little with the free play of the heart; or of short, close, firm, and strong attachments. Again quoting Sibson's observations, the adhesions are generally longer at the apex than elsewhere; those over the left are longer than over the right ventricle; those over the auricular portion of the right ventricle are longer than those over its body and near the septum, and the same holds good in the case of the left ventricle. Over the right auricle they are much shorter than over the right ventricle. The attachments of the left auricle, the aorta, and the pulmonary artery are generally closer than those of the right auricle. In some cases the contiguous surfaces of the pericardium are agglutinated together, the sac being entirely obliterated; and when this condition is of old standing, separation of the two surfaces is impossible without tearing the heart substance. Occasionally, when comparatively recent, they may with care be drawn asunder; or firm adhesions of old standing may exist side by side with those of recent origin, the result of a fatal intercurrent acute pericarditis, which can be easily broken down. The degree of thickening differs a good deal, but it may be very remarkable, as much as a quarter to half an inch or more; it chiefly affects the visceral layer. The heart is then enclosed in a dense, strong, tight envelope or casing, which compresses and strangles the organ in its grip.

(*c*) There is a distinct class of cases in which the adhesions are entirely external or *exo-pericardial*, the outer surface of the pericardium being more or less extensively fixed to the front of the chest, and often to the pleuræ, while the internal surfaces are quite free. They are usually chronic in their course, and secondary to neighbouring morbid conditions; they are especially met with in association with very chronic phthisis. These exo-pericardial adhesions may, however, extend from similar pleuritic changes, or may possibly result from a mediastinitis occurring at the same time as the attack of pleurisy which led to the pleural lesions. I have comparatively recently had under my care a case in which pericardial and pleuritic adhesions were diagnosed, associated with extreme double mitral disease and much enlarged heart; and except that the pericardial adhesion was entirely external, the diagnosis proved to be correct. The condition now under consideration is really mediastinal, and has been named *chronic mediastinitis* (*vide* "Diseases of the Mediastinum," vol. vi.)

(*d*) The most serious group of cases of pericardial adhesion are those which are both internal and external, there being a general matting of the sac to the heart, as well as to the chest wall in front, to the adjacent pleuræ, especially the left, to the diaphragm more extensively than in health, and occasionally to the structures in the posterior mediastinum and the spinal column. As a rule these conditions are accompanied with much thickening. When there is little or no general mediastinitis the term *pericarditis externa et interna* is applied; when there is a considerable increase of fibrous tissue in the mediastinum the condition is known as *indurative mediastino-pericarditis.* [These changes are more fully dealt with under "Diseases of the Mediastinum," vol. vi.] The external adhesions vary considerably in area, but in extreme cases may extend from the second cartilage to the sixth; from the manubrium to the upper half of the ensiform cartilage; and from the right border of the sternum to the apex of the heart to the left of the nipple line (Sibson).

(*e*) Exceptional instances are met with in which the prominent change is marked thickening of the pericardium, especially of its visceral portion, with little or no adhesion of the surfaces; and there may even be more or less fluid incarcerated between them. It is important to bear this variety in mind, for it may produce very serious effects upon the heart, with the consequent symptoms, without giving rise to any of the physical signs of pericardial adhesion. A very striking illustrative example was under my care not long ago in University Hospital.

Effects upon the heart and great vessels.—There has been much controversy as to the effects of pericardial adhesions upon the heart; they may vary much, of course, under different circumstances. In a considerable proportion of cases the organ is unaffected, either functionally or structurally, and, provided it be free from valvular disease, remains of its normal size. The obvious tendency is to embarrass its action more or less; the embarrassment is greater in proportion to the extent and firmness of the adhesions, and greatest when they are both internal and external.

One of the most frequent and important structural changes affecting the heart which may result from adherent pericardium is enlargement of the organ. Hope maintained that this morbid condition always gave rise some cases, but I cannot think that it represents the usual course of events.

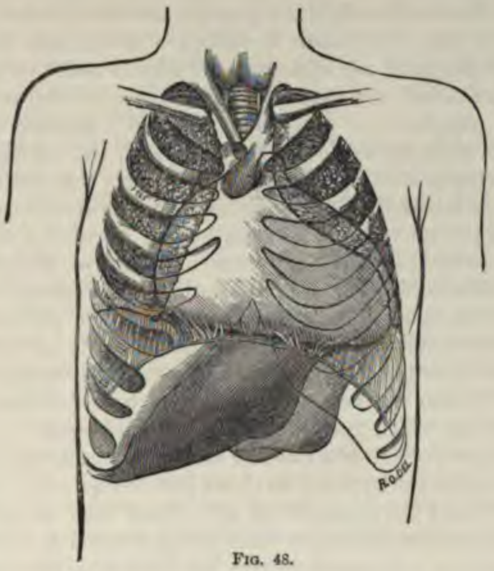

FIG. 48.

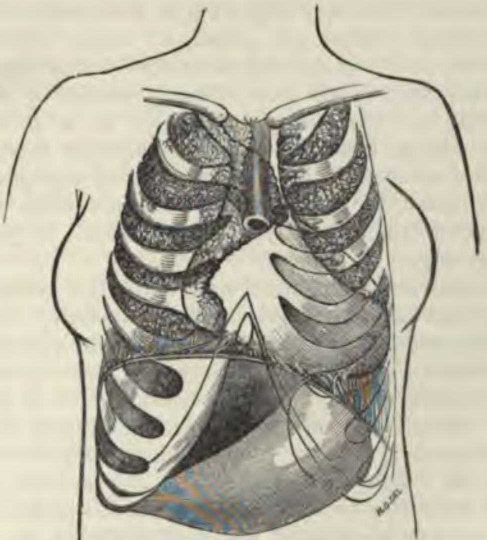

FIG. 49.

Figures showing position of internal organs in cases of adherent pericardium. (Sibson.)

As regards the nature, extent, and degree of the cardiac enlargement, considerable differences are observed in different cases of simple pericardial adhesion. As a rule there is a combination of hypertrophy and dilatation, the latter commonly preponderating; and it may exist practically alone. Both sides of the organ are usually involved more or less; but I fully accept Dr. John Broadbent's statement that pericardial adhesions in themselves are much more likely to affect seriously the right ventricle than the left, for reasons which he has pointed out in his monograph. The auricles are much less affected; indeed it may happen that, while the right ventricle is much enlarged, the auricle is compressed and may even be practically obliterated. When the enlargement of the heart is associated with valvular disease, it will necessarily be influenced chiefly by the nature of such disease, but in particular instances it may certainly be modified by the adhesions. In some of these combined conditions, with firm adhesions, Sibson described the ventricles as undergoing a change in form, becoming flattened out, the right in front of the left, and the septum flattened instead of bulging forwards into the right cavity. As a result of dilatation produced by adherent pericardium, and involving the orifices, valvular incompetence is prone to follow, especially at the tricuspid opening, which may become greatly enlarged.

In a small proportion of cases the effects of pericardial adhesions upon the heart are quite the opposite to those just considered. In children the natural growth and development of the organ may be prevented; or it becomes small and atrophied, its walls being grasped and compressed, and its cavities forcibly contracted in size by the dense, thick, tight envelope surrounding them. This may happen also from mere thickening of the visceral pericardium, without any adhesion. Other cardiac changes apt to occur in these conditions are degenerations—either fatty, pigmentary, or fibroid. They may result from direct pressure, or pressure on the coronary vessels; or the last may be due to a chronic interstitial myocarditis spreading from the pericardium. In some instances, no doubt, these cardiac changes are the outcome of myocarditis associated with an acute attack of pericarditis. They are often of considerable importance, and contribute largely to the symptoms of pericardial adhesions and thickening.

When the pericardium is fixed externally, the great vessels at the base of the heart are often abnormally exposed. Sibson observed that with enlargement of the heart "the great arteries are lifted up on the top of the ventricles into an unusually high position, and are crowded into the narrow space at the top of the chest, almost as high as the root of the neck." Occasionally one or both are compressed or constricted by pericardial adhesions; or their walls undergo degenerative or fibroid changes. As the result of obstruction to the general venous circulation, produced indirectly by adherent or thickened pericardium, the large veins become more or less dilated, and such dilatation may ultimately be extreme.

Clinical history.—It is obviously impossible to give any definite clinical description that will apply even to the majority of cases of adherent pericardium; all I can do will be to point out the symptoms and physical signs which may be associated with this condition, as well as the relations of these phenomena to each other, upon which a diagnosis may reasonably be founded. They vary considerably in individual instances, not only in respect of the actual nature and degree of the changes affecting the pericardium, but also of their effects upon the heart, and their association with endocardial lesions, with vascular diseases, or with neighbouring morbid conditions.

As was stated in the introduction to this subject, a large number of cases of pericardial adhesion do not exhibit any symptoms or physical signs whatever; and, unless there happen to be a well-known history of acute pericarditis, the condition cannot even be suspected during life. This applies not only to partial and loose adhesions, which often do not disturb the heart in any way, but even to cases in which there is general agglutination of the internal surfaces; provided the organ itself be not materially damaged. It is well to bear in mind the possibility of this condition, if with acute pulmonary inflammatory affections the heart should exhibit signs of embarrassment quite out of proportion to their severity. My observations have led me to the conclusion that it may add seriously to the danger under these circumstances, and even account for an unexpected death. The more pronounced the pericardial changes, the more prominent and definite are the clinical phenomena likely to be; and they are especially well marked when there is much thickening, and when the adhesions are both external and internal.

The symptoms and physical signs which may be met with will now be considered separately.

That pericardial adhesions may be the cause of *pain*—of painful, dragging, or other unpleasant sensations over the precordial region, I have not the slightest doubt, and when in cases of obvious chronic cardiac disease such sensations are much complained of, their existence may be reasonably suspected, and they should be carefully looked for. I have met with not a few instances in which they were associated with adhesions easily demonstrable on physical examination. Moreover, the pain occasionally comes on in attacks of an anginal character, when the case, if accompanied by other symptoms characteristic of such attacks, may present a perilous aspect. A feeling of precordial oppression, and inability to take a deep breath, are sometimes prominent symptoms, especially when the external adhesions are extensive. The patient is usually conscious of the disturbances of cardiac action associated with adherent pericardium, and is then likely to complain of palpitation, even at rest, but especially after exertion; and this symptom is sometimes very prominent.

Adherent pericardium ought always to be thought of as a possible cause of *palpitation*. The heart's action is in some instances irregular or unequal, and it may be so embarrassed as to lead to faintness or actually to syncope. The persistence of rapid cardiac action, in spite of treatment, may be important evidence of the formation of pericardial adhesions in children and young persons.

Pericardial adhesions may themselves unquestionably cause *dyspnœa* on exertion, sometimes well marked; and thus also they often add to the difficulties of other cardiac affections. No other respiratory symptoms can be definitely attributed to these conditions alone; but when there is much thickening, with compression of the heart and changes in its walls, the pulmonary circulation is likely to be embarrassed, and cough, expectoration, or even hæmoptysis to set in.

A very important and prominent group of symptoms in certain cases of pericardial adhesion are those indicating serious hampering, or actual failure of the right ventricle, and consequent interference with the general venous circulation. These either come on gradually, becoming more and more pronounced; or, occasionally, they supervene with great rapidity, the ventricle appearing to break down and give way very speedily, or even suddenly. They occur not only in cases where this cavity is obviously dilated, but also where the heart is strangled and compressed by dense fibrous thickening; and in such cases they may be extreme. No doubt they depend in great part upon the associated changes in the cardiac structure. These symptoms are similar to those which arise in other forms of heart disease affecting the right side; namely, *general dropsy*, involving the serous cavities as well as the subcutaneous tissue more or less extensively, congestion of the hepatic and portal system and its consequences, and also of the kidneys, nervous system, and other structures. The dropsy usually begins in the legs, but it may ultimately involve the trunk, and even the arms. In exceptional instances ascites is noticed before anasarca. Remarkable cases occasionally occur, entirely due to pericardial adhesions and their consequences, in which the peritoneal cavity and pleuræ become repeatedly full of fluid, and have to be tapped again and again in order to afford temporary relief. Under these circumstances the breathing is likely to be much distressed, even to the degree of orthopnœa. The appearance of the patient differs in different cases. *Cyanosis* with distended veins may be evident; or, on the other hand, there is sometimes marked pallor, with puffiness of the face. The *liver* becomes enlarged so that it can readily be felt below the ribs, and may be painful and tender. Occasionally it reaches even below the umbilicus, appearing to be very large; but then it is usually displaced downwards as well: after a time the organ yields an abnormally firm sensation on palpation, and may become irregular; in prolonged cases it may even pulsate. Symptoms connected with the *alimentary canal* are often prominent, and sickness may be troublesome. The *spleen* is sometimes perceptibly enlarged. The *urine* is more or less diminished in quantity, concentrated, and often albuminous. I have known the amount of albumin to be so large that the urine became almost solid on boiling, simulating serious renal disease. In bad cases the patient is very restless and sleepless.

Dr. John Broadbent, speaking of the symptoms which have just been discussed, deduces from his observations the following corollary:—"That when symptoms of right ventricle failure supervene in cases in which there is no evidence of left ventricle failure due to valvular disease or kidney mischief, constant high tension, or other obvious causes, or of lung disease such as chronic bronchitis, etc., to account for their appearance, the presence of adherent pericardium should be suspected as the cause, and other indications of it carefully sought for. So, too, in valvular disease of the left ventricle, in which the lesion is judged to be slight, and compensation breaks down unaccountably, adherent pericardium should be thought of." Further, speaking of the difference in the symptoms of right ventricle failure when due to pericardial adhesions, and when secondary to valvular disease of the left ventricle, he writes: "In cases of right ventricle failure attributable to adherent pericardium, there is no cyanosis, though the respirations may be hurried, and there may be some dyspnœa; there may be an entire absence of dyspnœa, though the other symptoms are severe; there is usually no congestion or œdema of the lungs. The dyspnœa, when present, is probably due to deficient supply of blood to the lungs and a feeble pulmonary circulation owing to the failing powers of the right ventricle." While fully recognising the correctness of these conclusions in their application to a certain class of cases, I must point out that they by no means always hold good in relation to adherent pericardium; for the effects of the difficulties in the right side of the heart may themselves lead to cyanosis and dyspnœa, while the lungs may be also implicated when the entire heart is gripped by strong adhesions. Moreover, pericardial adhesions may help in producing this class of symptoms in cases where there is pronounced valvular disease on the left side.

Physical signs.—The existence of pericardial adhesion can often be recognised positively and demonstrated by careful and systematic physical examination; and it is most desirable to have a clear and definite knowledge of the signs which, in different combinations, have to be looked for and studied in respect of this condition. At the same time it must be understood that they are frequently absent, or at any rate not at all characteristic; and this may happen even when there are very pronounced symptoms directly due to an adherent pericardium; for example, if the heart is compressed and atrophied, though the symptoms may be extreme, the signs will be wholly indefinite. They are likely to be better marked as the adhesions are more extensive and dense, and especially when these are external as well as internal. They result not only from these lesions themselves, but also from their effects upon the heart and vessels, and upon the circulation. They may be considered in the following order:—

(i.) *Change in shape.*—In exceptional instances a distinct and permanent depression of more or less of the precordial region, with narrowing of the intercostal spaces, is observed; the structures being drawn in by thick external adhesions. Far more commonly, however, there is abnormal fulness or bulging, due to enlargement of the heart; but as this usually depends mainly on other causes, it can hardly be regarded as an indication of adherent pericardium, except under particular circumstances.

(ii.) *Signs associated with cardiac movements.*—Certain visible and tactile signs coming under this head are of the utmost importance, and demand somewhat detailed consideration. Sometimes there are peculiarities in the cardiac movements which cannot well be described, but which are very suggestive of these changes, when prominent cardiac symptoms are present, and are not obviously due to any other organic affection of the heart. The following are the more definite signs to be studied :—

(*a*) *Apex-beat.*—In cases of adherent pericardium, the ordinary apex-beat presents many differences as regards its position, force, and characters; but these depend mainly upon the effects of the particular valvular disease or diseases with which the condition happens to be associated. Thus it has been noticed far to the left, and presenting all the indications of a greatly hypertrophied left ventricle. One of the signs to be looked for is a displacement of the apex-beat, which is fixed in its abnormal position, and cannot be modified by any change of posture. As a rule it is carried somewhat outwards; but the most suggestive displacement is elevation, it may be to the fourth space or even higher, while perhaps at the same time there may be marked evidence of hypertrophy. In many instances the apex-beat is very feeble, or even imperceptible when other phenomena, to be presently described, are well marked; and even when it extends 1 to 1¼ inch outside the left nipple. This is attributed to small size and weak action of the heart; to restraint of the organ by adhesions; or to much thickening of the pericardium. When it is wholly due to feeble cardiac action, the beat may at times be perceptible, at other times not. There may, however, be a distinct impulse over the ensiform cartilage or in the epigastrium.

(*b*) *Impulse.*—Taking into account the entire impulse, it must be admitted that in cases of adherent pericardium great variation of its situation, extent, force, and characters is observed; but there are certain points deserving of attention. A remarkable extension of its area is often noticed, especially upwards over the precordial region; and it may reach the second space or cartilage. This may be associated with obvious elevation and fixation of the apex; or it may be impossible to localise any definite apex-beat. At the same time the impulse is often strong and superficial, the heart pulsating in close contact with the chest walls. In some instances the movement presents to the eye a decidedly undulatory or wave-like character, from the base towards the apex. In others it is peculiarly jarring, or has an abrupt jogging quality. The rhythm of the cardiac action is sometimes markedly disturbed, and pericardial adhesions may undoubtedly give rise to irregularity. When the heart is at the same time enlarged, the extent of the impulse is correspondingly increased, often passing considerably beyond its normal limits, and probably tending more towards the right, in consequence of the greater enlargement of the right ventricle.

(*c*) *Systolic recession or retraction.*—A visible recession or retraction of certain parts of the chest wall, associated with the ventricular systole, has attracted much attention in respect of adherent pericardium. There can be no doubt that the signs coming under this head are of great importance in the diagnosis of this condition, and they deserve particular study in any suspected case. They come practically under three categories, namely :—

(α) Recession over the spot corresponding to the apex of the heart, occurring with or immediately after the systole. This phenomenon, when present, is usually associated with a definite apex-beat, but is sometimes noticed when there is no perceptible impulse at this point.

(β) Systolic depression of more or less of the precordial region, generally involving one or more of the intercostal spaces to the left of the sternum, especially the third, fourth, and fifth, along a variable extent of their length. The movement is sometimes distinctly wavy. In certain cases, where the adhesions are extensive and strong, and the heart is acting powerfully, the cartilages are also involved, or indeed even the lower half of the sternum, the ensiform cartilage, and the epigastrium. When the recession occurs simultaneously with an obvious and strong apex-beat, the combination is very striking, but it may be indefinite or absent. Should the right ventricle be greatly enlarged, a similar movement may possibly be visible in the intercostal spaces to the right of and close to the sternum ; of this I believe I have seen examples. According to Friedreich, the pitting is more marked at the height of inspiration.

(γ) Retraction of the posterior or lateral portions of the thoracic walls.—I cannot say that I am personally familiar with this sign, which, when present, is regarded by Dr. John Broadbent as a most important diagnostic sign of adherent pericardium ; he describes it in the following words :—"In cases of adherent pericardium, marked systolic retraction of some of the lower ribs on the lateral or posterior aspect of the thorax may sometimes be seen. This phenomenon is best seen when the patient is sitting up in a good light, and the movements of the chest are carefully observed from a short distance off, first from the front and then from the lateral aspect. When a pulsatile movement is seen over the lowest part of the left side posteriorly, it may at first sight appear to be expansile. On a more careful scrutiny it will be found that there is a tug on the false ribs during the cardiac systole, and a sharp rebound during diastole, which can be felt as well as seen when the hand is laid flat upon the chest wall at the spot ; it is more marked when a deep inspiration is made ; it may be seen occasionally not only on the left side but also on the right, especially if the patient leans over to the left."

Space will not permit of any long discussion of the associations of the phenomena just indicated with conditions other than pericardial adhesions, or of their precise significance in any individual case of such adhesions. A few general observations on these points must suffice. Apical recession very rarely occurs except as the result of adherent pericardium, but it was observed by Friedreich in a case of aortic stenosis where there were no adhesions ; and has also been noticed under other circumstances. When it is associated with a definite beat it probably indicates that the apex of the heart is fixed to the chest wall, and drags on it during the systole. The adhesion need not, however, be extensive, for a narrow band may cause the depression, provided the pericardium be fixed externally. When there is no palpable apex-beat, it is supposed that the heart is prevented by adhesion to the diaphragm or vertebral column from performing its normal forward and rotatory movement during systole; or that the cardiac impulse is too feeble to be felt through the adhesion.

Skoda was of opinion that systolic recession of the intercostal spaces is pathognomonic of adherent pericardium, but numerous observations have shown that this is not the case, as the phenomenon may occur in cases of considerably enlarged heart, as the result of atmospheric pressure, especially when associated with aortic regurgitation. Still it is an important sign of adhesion, and its presence should always have due weight in diagnosis. As a rule it indicates that the contiguous surfaces of the pericardium are adherent, and also that the sac is fixed in front to the chest wall, and to some structures posteriorly, so that when the heart contracts, being firmly attached behind, it pulls in more or less of the yielding anterior thoracic wall. When there is no posterior adhesion, and yet systolic depression occurs, it is supposed that the firm attachment of the pericardium to the central tendon of the diaphragm forms the fixed point from which the heart acts in drawing in the front of the chest, or possibly that the effect may be produced by the contraction of the organ itself. Friedreich is of opinion that the lower surface of the heart must be firmly adherent to the diaphragm. I have met with this phenomenon in a pronounced form in cases of external pericardial adhesion with enlarged heart, where the internal surfaces of the sac were quite free. As a result of diminution in the force of the cardiac action, a marked systolic retraction may in course of time become less and less evident, and finally disappear.

The systolic retraction of the posterior or lateral portions of the thoracic walls, which, as we have seen, is regarded as a positive sign of adherent pericardium by Dr. John Broadbent, who states that it is quite distinct from recession of the lower ribs in inspiration, is explained by him in the following way:—"The heart is, by means of the pericardium, adherent not only to the central tendon of the diaphragm, but probably also to a large area of the fleshy or muscular portion of the diaphragm, and, it may be, to the anterior thoracic wall as well; as it contracts it drags upwards and inwards the less resistant fleshy part of the diaphragm towards the central tendon or anterior chest wall; hence the points of attachment of the digitations of the diaphragm to the lower ribs and costal cartilages are dragged inwards and downwards. It will always be found in such cases that the retracted portions of the chest wall correspond to the floating ribs or costal cartilages of the lower ribs at the points of attachment of the diaphragm."

(*d*) *Diastolic shock or concussion.*—This is a very exceptional sign, only occurring where the pericardium is firmly adherent to the anterior chest wall, and when the heart is acting powerfully. It follows immediately after the systolic recession, and is in proportion to its force. The diastolic shock is felt by the hand as a "back stroke." It may be perceptible only at the apex-beat, over one or more intercostal spaces, over a more extensive surface—possibly over the entire precordial area; or even round the left side to the back. The phenomenon is attributed to the elastic recoil or rebound of the chest wall, at the beginning of diastole as soon as the systolic dragging force has ceased. In well-marked cases it may be felt as a distinct jerk or blow, which is occasionally so strong as to be like the impulse of the heart. When present it is regarded as a pathognomonic sign of adherent pericardium.

Apart from the sign just considered, I feel sure that in some cases of adherent pericardium, with exposure of the heart and great vessels, a diastolic impulse is felt, due to the closure of the aortic and pulmonary valves. It is noticed over the base, and is quite independent of systolic retraction.

(*e*) *Posterior systolic impulse.*—I believe that this sign is sometimes of value in the diagnosis of adhesion of the pericardium to the structures posteriorly; especially when there are indications of probable agglutination of its two surfaces, and of anterior adhesions. It is best recognised, not by the hand, but by the head, when this is placed over the back of the left side of the chest in the practice of direct auscultation. The movement is directly due to the hypertrophied heart, and is often associated with more or less compression of the lung, which therefore conducts the sensation more readily; but I think that it is likely to be more pronounced when the structures are matted together by adhesions.

(iii.) *Cardiac dulness.*—Pericardial adhesions or thickening do not in themselves appreciably affect the cardiac dulness, as a rule; but a mass of fibrous tissue about the vessels may certainly cause some increased dulness towards the base. When, as a consequence of adhesions to the chest wall, the heart and great vessels are abnormally exposed and superficial, the area of cardiac dulness will be proportionately enlarged, and may be of considerable extent, being often markedly increased in an upward direction, sometimes reaching the second rib. Part of this altered percussion sound may be due to adhesion and collapse of overlapping lung. When enlargement of the heart is associated with the pericardial condition the dulness will be modified accordingly, and is not uncommonly very extensive. Dr. John Broadbent writes: "When, during an attack of pericarditis, the area of cardiac dulness has been noted to increase considerably in extent, and after the subsidence of the attack remains permanently increased, it is extremely probable that adhesions have taken place, fixing the heart in a condition of dilatation." In well-marked cases the dulness resulting directly or indirectly from pericardial adhesions and thickening is very pronounced or even absolute. As already stated, when extensive calcification has taken place, the percussion sound in rare instances has been described as presenting a peculiar osteal quality.

(iv.) *Auscultatory signs.*—It cannot be said that there are any actually pathognomonic or trustworthy auscultatory signs of adherent pericardium; but one or other of the following points may be worthy of attention in particular cases :—

(*a*) Should the pericardium be fixed to the chest wall the heart sounds are likely to be remarkably superficial. The first sound is certainly often abnormal in character. In some cases it is peculiarly sharp and valvular in quality; in others it is markedly dull or muffled at the apex or over the mid-cardiac region; or again it may be prolonged and reduplicated. The second sound is frequently reduplicated, but Friedreich maintains that this may be due to the rebound of the chest wall which causes the diastolic shock, and produces a dull sound heard after the second sound of the heart. Dr. John Broadbent regards a weak pulmonary second sound, when there is evidence of hypertrophy of the right ventricle, as a very important indication that the cause of the hypertrophy was probably not back pressure through the lungs due to left ventricle trouble, but some intrinsic cause, perhaps adherent pericardium. Marked conduction of the heart sounds towards the back of the left side of the chest, especially when associated with the feeling of pulsation already referred to, is suggestive of posterior pericardial adhesion.

(*b*) A rough pericardial friction-sound may remain over different points of the precordial region, especially towards the base, for some time after an attack of pericarditis; and, should it be associated with suspicious signs of adhesion, might be useful as corroborative evidence. Its eventual disappearance would probably indicate that adhesions had formed at the spots where it was previously audible, and have since become more or less general.

(*c*) With regard to endocardial murmurs, a kind of rumbling presystolic murmur is sometimes heard at the apex, which does not, however, indicate the presence of mitral stenosis; this kind of presystolic murmur is specially common in children (J. Broadbent). It is possible that a basic systolic murmur may result from the pressure of pericardial thickening upon one or both of the great arteries. The several valvular diseases, when present, will give rise to their corresponding murmurs, but I believe that these may be modified in their character by adherent pericardium, and a tricuspid regurgitant murmur may ultimately result from enlargement of the right ventricle owing its origin to this condition.

(v.) *Signs connected with respiratory movements.*—When searching for pericardial adhesions, it is often highly advantageous to study the effects of deep inspiration and expiration. In the first place, the fact that the position of the apex-beat and the area of extended cardiac impulse are not thus affected may be of much importance; as well as that the area of precordial dulness is not altered. It implies the presence of adhesions between the external surface of the pericardium and the thoracic wall, and the want of any modification in the dulness is particularly marked when the sac is adherent to the margins of the lungs also. As a result of extensive external pericardial adhesions, inspiratory expansion may be decidedly less on the left than on the right side. Another sign sometimes observed, coming under this head, is impeded descent of the left half of the diaphragm in inspiration, as indicated by diminished movement of the upper part of the abdominal wall on that side. This may occur either with or without adhesion to the anterior chest wall; in the latter case it has been attributed to abnormal attachment of the pericardium to the muscular portion of the diaphragm, which hinders its descent. Tracheal tugging might possibly result from adhesion of the pericardial sac to the bifurcation of the trachea, but I have never found this to be the case.

(vi.) *Arterial signs.*—In some cases in which pericardial adhesions were proved after death to exist, I have observed a peculiar visible movement, in connection with the large arteries at the root of the neck, which I believe may be of more or less significance. It gives the impression that the heart is making an effort to drive the blood into these vessels, but is prevented from doing so effectually on account of the embarrassed action due to the adhesions. The movement may be modified by the coexistence of aortic or mitral disease. Irregular pulse may be associated with adherent pericardium, but commonly this is not the case until cardiac failure sets in; and in diagnosis no positive reliance can be placed on this disturbance. I think that in cases of mitral disease the condition tends to increase the irregularity. The arterial sign to which Kussmaul attached special importance is the presence of a marked *pulsus paradoxus*, the pulse intermitting with inspiration, which has been chiefly noticed in cases of indurative mediastino-pericarditis, but occurs under other circumstances, and is by no means trustworthy. Kussmaul attributes it to the presence of fibrous cords encircling the aorta, which, by dragging on it during inspiration, constrict its lumen.

(vii.) *Venous signs.*—Sudden collapse of the veins of the neck during the ventricular diastole has been specially studied by Friedreich, who regards it, when associated with systolic retraction of the intercostal spaces, as a most valuable sign of adherent pericardium; it is never present in any striking degree without such retraction. The veins, often tensely filled during systole, disappear from view during diastole, the subsidence being synchronous with the diastolic shock felt in connection with the chest wall. Sometimes the supraclavicular fossæ are deepened at the same time. The explanation of this phenomenon given by Friedreich is that, owing to the diminution of the thoracic space, the return of blood through the cervical veins is hindered during systole; and that the subsequent sudden diastolic enlargement has an aspiratory effect, drawing in the blood from the veins: it is supposed also that the diastole takes place with unusual force and rapidity, owing to traction by the adhesions from without, and the descent of the raised diaphragm. He further assumes that in consequence of the diastolic descent of the heart, especially as caused by the action of the diaphragm, the large vascular trunks, including the superior vena cava, become elongated, and thus the downward current of blood from the cervical veins is hastened.

Dr. John Broadbent quotes a case of adherent pericardium, observ by François Franck, in which systolic emptying of the veins of the ne occurred, and was ascribed to an aspiratory periventricular effect caus by the adhesions. He also describes another case—where the pericardiu was universally adherent to a greatly hypertrophied heart, and also the chest wall over a large area—in which systolic emptying of enlarged vein on the front of the chest, to the right of the sternu was followed by filling during diastole. "The explanation suggeste was that the pericardium adherent to the heart and chest wall dragge apart the walls of the internal mammary vein during systole, causin a suction action, so that the blood was drawn into its lumen from t afferent veins during systole." I think I have recently met with a simil case.

Diagnosis.—It must be repeated that in a large proportion of cas where pericardial adhesions exist, there are no trustworthy data upo which a definite diagnosis can be based; though nevertheless the possibilit of their existence may suggest itself in explanation of cardiac disturban of obscure origin. The rule is not to forget these lesions in any cas and to take some trouble in their clinical investigation. In not a fe instances the diagnosis of adherent pericardium is evident enough, an yet the condition is entirely overlooked. It is not enough to say th pericardial adhesions exist; an endeavour must be made to determin their extent and nature; whether they are external, internal, or both and their effects upon the heart. Moreover, their association wit valvular diseases of this organ must not be lost sight of, as they are ofte important factors in such combinations.

If the patient have had one or more attacks of acute or subacut pericarditis, or of rheumatic fever, we may suspect adhesions; especiall if they have formed under the observation of the practitioner who ha subsequent charge of the case, and who can give definite information a first hand. I have not uncommonly watched their formation durin the period of convalescence, and had the opportunity of studying thei after-effects. In other instances an indefinite history merely poin to cardiac inflammation of some kind. The frequent association of per carditis and endocarditis in childhood has an important bearing o diagnosis; and, when the origin of valvular disease can be traced t early life, pericardial adhesions should be particularly looked for. Un fortunately, in a large proportion of cases no history pointing t pericarditis can be obtained; and it must not be forgotten that th formation of adhesions may be a chronic process throughout.

The positive diagnosis of adherent pericardium is founded upo careful and systematic investigation and study of the symptoms an physical signs already discussed, not only in themselves, but also i relation to each other. Individual cases differ much in their exac characters. Sometimes the diagnosis has to be made on physical sign alone, there being no prominent symptoms. On the other hand progressive signs of general venous obstruction following an attack o pericarditis, inducing extreme dropsy of the subcutaneous and serous cavities, only to be relieved by repeated operations, may alone indicate the presence of a thick, dense, adherent pericardium, compressing the heart, there being no obvious physical signs of the condition. In other instances, again, enlargement of the heart, especially of the right ventricle, occurring without other adequate cause, or perhaps developing with unusual and inexplicable rapidity in connection with valvular disease, suggests adherent pericardium as a possible cause. With regard to the relation of symptoms to physical signs, Dr. John Broadbent writes: "When symptoms of cardiac failure, more especially of right ventricle failure, occur of greater severity than the physical signs present seem to warrant, or where compensation breaks down unaccountably, adherent pericardium must be suspected. When rest and suitable treatment fail to give relief, provided the patient is not of advanced age or thoroughly broken down, this affords further evidence in favour of adherent pericardium, and other confirmatory signs of it should be carefully looked for." With these remarks I cordially agree. Sir Samuel Wilks has expressed the opinion that severe heart symptoms in young persons, without valvular murmurs, point to pericardial adhesions; while in persons of mature age they indicate cardiac degeneration. There is a good deal of truth in this statement, though not a few exceptions will be met with in both directions.

Prognosis.—No general rules of practical value can be stated under this head; every case in which pericardial adhesions exist must be studied individually as regards prognosis. Often they are of no consequence whatever; in other instances they are merely a source of discomfort, and do not endanger life. Sometimes, however, they are extremely grave in themselves, and then the outlook is very serious, while they make life exceedingly miserable. It may be impossible to give any relief to the symptoms; or a dropsical condition may be kept at bay only by repeated operations. That pericardial adhesions add seriously to the effects and dangers of valvular diseases cannot be doubted, and they often hasten their progress and fatal termination.

Treatment.—Pericardial adhesions once formed cannot be got rid of. Rest, good nourishment, and other suitable measures are of value in preventing or delaying their ill-effects, and in maintaining the nutrition of the myocardium. Whether the various exercises now in vogue in the treatment of cardiac disease are likely to be of any service in this kind I do not know, but in cases where extensive and firm adhesions exist they certainly may do much mischief, if carried out thoughtlessly. Cardiac tonics may be useful in some cases; but it must be remembered that pericardial adhesions may materially interfere with the action of digitalis and allied agents upon the heart, and then such agents may do much more harm than good. Symptoms must be dealt with on ordinary principles; and dropsy often requires repeated removal by operation.

IV. HYDROPERICARDIUM; DROPSY OF THE PERICARDIUM

Pathology and Etiology.—Hydropericardium, or hydrops pericardii, signifies a serous effusion into the pericardial sac, occurring during life, of a dropsical nature, as distinguished from one of inflammatory origin. As has been previously stated, a certain quantity of fluid, varying under different circumstances, is found in this sac at most necropsies; this is merely due to transudation from the vessels and heart occurring during the act of dying, and for a time after death. It usually amounts to from half an ounce to an ounce, but under favourable conditions may reach three ounces or more. Definite hydropericardium may occur under the following circumstances:—(i.) As an acute or active effusion in connection with certain cases of Bright's disease, thus it may follow scarlatina. (ii.) As a part of chronic dropsy, more or less general, usually in cases of cardiac or renal disease; but occasionally associated with scurvy and allied states, grave forms of anæmia, tuberculosis, cancer, and other cachexiæ affecting the blood. In this group the pericardial dropsy almost always follows effusion into the pleuræ, and the pericardium is much less frequently involved than other serous membranes. (iii.) Exceptionally from some mechanical difficulty interfering with the local circulation. It may thus occur in connection with certain affections of the lungs, or even of the heart itself, impeding the return of blood from the cardiac and pericardial veins; and with disease or thrombosis of these veins, atheroma of the coronary arteries, aneurysm, chronic mediastinitis, or a mediastinal tumour causing pressure upon the veins. Hydropericardium has been known to follow sudden extreme pneumothorax.

Dr. W. Ewart (20) has drawn special attention to cases of latent and transient pericardial effusions, which may occur, independently of acute pericarditis, under the influence of rheumatism, of cardiac affections, of Bright's disease, and so forth. He considers that they may be dependent upon a subacute inflammatory process, but that probably they are more often passive or mechanically induced. No doubt such cases are met with, and if the fluid be rapidly reabsorbed they may run their course entirely unobserved.

Anatomical characters.—The essential morbid condition in hydropericardium is the presence of a quantity of serous fluid in the sac, which has collected during life, but which is not accompanied by any indications of inflammation. The amount varies considerably in different cases. In the large majority of instances it is moderate, from six or eight to twelve ounces; but it certainly may reach a pint to a pint and a half; as much as four pints have been reported, though it is very doubtful whether such large effusions are not really of inflammatory origin. The fluid is, as a rule, clear, and either colourless or of a yellowish or greenish tint. It is sometimes turbid from admixture of degenerated epithelium, or may be tinged with blood pigment or bile. Hæmoglobin may, however, have escaped after death. The effusion is alkaline; and in composition resembles more or less the serum of the blood, with differences in the relative proportion of the albumin and other constituents. Even a dropsical accumulation in the pericardium may be spontaneously coagulable. In renal cases it may contain urea. When the fluid is abundant it tends to produce, in proportion to its amount, the physical effects upon the sac itself, the heart, and neighbouring structures already discussed under inflammatory effusion. In prolonged cases the pericardium may become sodden, its epithelium being also changed; and it is said that the subserous tissue about the heart loses its fat and becomes œdematous. Possibly, moreover, the pressure of the fluid in course of time may impair the nutrition of the myocardium. In the majority of cases of hydropericardium, however, there is but little to be noticed beyond the presence of the fluid.

Clinical history.—The circumstances under which it occurs make it unlikely that there will be any definite symptoms of hydropericardium, especially if the fluid be but in small or moderate quantity. There is never any pain or other acute subjective sensation such as is met with in pericarditis. Should the effusion attain a large amount, it may certainly cause a feeling of weight and oppression across the chest, with precordial anxiety; and it will either induce or aggravate previous dyspnœa, obstruction of the venous circulation, and low arterial pressure, with the usual symptoms arising therefrom. In the large majority of cases it merely intensifies pre-existing symptoms, and it is often very difficult to determine the share of pericardial dropsy in their manifestation, though sometimes its effects are obvious enough, especially if it come on rapidly. It does not give rise to any febrile symptoms; and, as a rule, there is no particular disturbance of the heart's action.

It will thus be evident that by physical examination only can hydropericardium be positively recognised. The absence of friction phenomena, such as are associated with acute pericarditis, is a most important point of distinction between the two conditions. The signs of the effusion are similar to those fully described under pericarditis, to which the reader is referred. As a rule they only indicate the presence of a moderate amount of fluid, and there may be so little that it cannot be detected at all. It is affirmed that the dulness is more readily altered by changes of posture than in cases of inflammatory effusion. Hydropericardium generally follows effusion into both pleuræ; and the physical signs of this latter condition will probably be well marked before those of pericardial dropsy are revealed. The combination may also cause a difficulty in diagnosis. I have never met with a case in which acute pericarditis and hydropericardium could not be distinguished by due attention to the circumstances under which they severally occur, and to the points of distinction already indicated. Possibly in connection with Bright's disease an effusion might collect which it would be difficult to classify definitely as inflammatory or dropsical. As regards **diagnosis,** the chief danger is that hydropericardium is not thought of, and is consequently overlooked when physical examination would clearly have revealed it. The cases of latent and transient pericardial effusion referre to by Ewart must also be borne in mind, for it is probable that even whe considerable it is likely to be overlooked, unless accurate and searchin physical examination is made. Should the condition be associated with and secondary to certain local affections within the chest, the diagnosi may be very obscure and difficult. The **prognosis** in cases of pronounce dropsy of the pericardium is, for obvious reasons, usually very grave, an it generally indicates a speedily fatal termination. Temporary improve ment or even recovery may, however, take place in some instances unde favourable conditions.

Treatment.—As a rule treatment has to be directed to the cause o the hydropericardium, and the measures persisted in which have bee previously carried out for the relief of the general dropsy which it usuall complicates. It might be desirable in some instances to relieve th venous circulation by venesection or local removal of blood. Cardia tonics are to be used when required. The application of blisters has bee found advantageous occasionally in promoting the absorption of pericardia dropsy. Whether tapping is permissible or desirable can only be deter mined by a careful consideration of the circumstances of each individua case.

V. HÆMO- OR HÆMATO-PERICARDIUM; BLOOD IN THE PERICARDIUM

Etiology.—It is not uncommon to find a certain amount of bloo mixed with inflammatory products in the pericardium; but the circum stances under which pericardial hæmorrhage may occur as an independen condition are as follows:—(i.) As a consequence of traumatic injury fro without, or by foreign bodies penetrating from the œsophagus. (ii. Associated with scurvy, purpura, or, extremely rarely, leucocythæmia an allied conditions. (iii.) From rupture of the heart or of a cardiac aneurysm (iv.) From lesions of the aorta. An aneurysm of the first part of th arch is very apt to open into the pericardium, not uncommonly b a pin-hole rupture. Rarely this event happens in the case of aneurysn of the descending aorta; and in one reported by Dr. Herbert Habersho the aneurysm was situated at the junction of the transverse an descending portions of the arch of the aorta. A case is reported b Dr. Charlewood Turner in which rupture of the inner coats of th aorta was followed by a dissecting aneurysm, which perforated int the pericardial sac. Dr. Rolleston has described a very interestin condition (39) where the inner and middle coats of the commencement o the aorta ruptured transversely, and the blood leaked into the pericardium through a small hole the size of a pin's head in the external coat; bu there was no dissecting aneurysm. (v.) From rupture of smaller vessels namely, one of the coronary arteries, especially if it be the seat o aneurysm; or of vessels in a new growth.

Anatomical characters.—The quantity of blood which collects in th pericardial sac varies under different circumstances. When there is large opening and rapid extravasation takes place, it is much less than when it escapes gradually through a small aperture. When an aneurysm bursts freely into the pericardium, the quantity usually found is said to be about 7 ounces, whereas in the case recorded by Dr. Rolleston already referred to it amounted to over 24 ounces. A traumatic case has recently been reported by Dr. Mansell Moullin in which over 6 pints of thin dark fluid blood were removed from the pericardium in the course of three hours. The patient recovered. The blood may appear as a soft red clot, jelly-like, or more or less decolorised; while a variable and sometimes considerable amount of serum will probably have separated from it. In Dr. Habershon's case the pericardium contained about a pint and a half of dark fluid blood. Hæmorrhage in the pericardium may set up pericarditis. The sac is distended in a proportionate degree when there is a large collection of blood in its interior.

Clinical history.—There may be previous symptoms or physical signs of the morbid condition which causes the pericardial hæmorrhage, but not uncommonly such is not the case, and the lesion is quite unexpected and sudden. Immediate or very rapid death usually occurs, but the event may be preceded by grave cardiac symptoms or collapse. In those cases where the accumulation takes place gradually, the patient may live some time, and may complain of pain, associated with serious cardiac disturbance, faintness or syncope, dyspnœa, and signs of loss of blood. The physical signs, if noted, will be those of an accumulation of fluid in the pericardial sac. (*Vide* Dr. Allbutt's case, p. 767.) The prognosis is hopeless as a rule.

Treatment, as a rule, can only be symptomatic. Stimulants and cardiac remedies may be of temporary service in the more prolonged cases. No operative interference is practicable in the great majority of cases, but Mansel Moullins' case, above referred to, is very suggestive as to what may be possible in some instances.

VI. PNEUMOPERICARDIUM AND ITS EFFECTS; GAS IN THE PERICARDIUM

Pneumopericardium is extremely rare, and it needs but brief consideration in this article.

Etiology.—Gas in the pericardium has been referred to the decomposition of fluid in the sac, especially if the fluid be of an ichorous nature; and it has even been said that this is its most frequent source. The probability is that such decomposition, in the large majority of cases if not always, is a post-mortem change. Its presence has also been attributed to secretion by the membrane, but on no adequate grounds. The two classes of cases in which it is clinically important are—(i.) *Traumatic*, from penetrating wounds, including paracentesis for effusion; fractured ribs; contusion or crushing of the chest, or injury from the side of the œsophagus. (ii.) *Perforative*, in which a communication is formed externally, or between the pericardium and a cavity or tube containing air. This kind of lesion has been already sufficiently described in relation to acute and suppurative pericarditis, and it will suffice to mention, as illustrations, perforation from the œsophagus, especially in connection with cancer; rupture into the pericardium of a phthisical cavity or pyopneumothorax; and perforation of a gastric ulcer. A remarkable case is on record in which a hepatic abscess communicated with the stomach and the pericardium, and thus air gained access to the latter. The entrance of gas into the sac may be aided by pressure, by the elastic traction of the lungs upon the pericardium, or by diminution of the size of the heart during systole.

Anatomical characters.—The gas in cases of pneumopericardium varies in its amount and composition, but it is generally offensive. It may so distend the sac, that when this is punctured the gas escapes with a hissing noise. Blood or other materials often gain an entrance at the same time as the gas; or at any rate inflammation is so speedily set up that pneumopericardium has never been clinically observed alone, fluid being always present, rarely serum—*hydropneumopericardium*—usually pus—*pyopneumopericardium;* or the fluid may be ichorous. In a case described by the late Dr. Begbie (9), yellow lymph was present on the surface, and a quantity of dark brown fœtid fluid in the sac. Whatever the position of the patient the gas will always be uppermost and the fluid below. The lungs will be pushed aside and compressed, and the diaphragm depressed, in proportion to the degree of distension of the pericardial sac.

Clinical history.—As might be anticipated, the symptoms of pneumopericardium and its consequences vary much in different cases, and are by no means characteristic. Sometimes there are none; or the patient is merely weak and apathetic. Should gas collect rapidly, there will probably be much precordial distress and sense of distension. The chief objective symptoms which have been observed in different cases are severe dyspnœa, cyanosis, fits of syncope, collapse, a feeble and irregular pulse, and rarely dysphagia. Sleep is necessarily disturbed, and delirium sometimes occurs. Occasionally pneumopericardium is accompanied with rigors, high fever, profuse sweats, and diarrhœa; but such symptoms are probably due to other and more general causes.

Physical signs.—It is upon the physical signs that the diagnosis of pneumopericardium and its consequences is practically founded; these being due to the presence of gas and fluid within the sac: most of them are very striking and peculiar. They may be briefly described as follows:—

(i.) The precordial region is likely to present abnormal fulness or bulging, which may be very pronounced.

(ii.) The apex-beat is weak or absent, but is better felt when the patient bends forwards. Sometimes an impulse is observed over several intercostal spaces.

(iii.) The cardiac movements occasionally bring out a very peculiar crackling sensation, due to the bursting of air-bubbles. Possibly a succussion-splash might be felt on shaking the patient.

(iv.) Percussion signs are usually very remarkable. Over the region corresponding to the distended pericardium there will be a tympanitic percussion sound, often with a pronounced metallic quality. It is said that a variation in its height, owing to alterations of the shape of the body of gas in the pericardium by the rhythm of the heart, may be detected by rapidly-repeated percussion. It has also been affirmed that the note differs in its degree of resonance during the systole and diastole respectively, the organ being situated farther forward and downward during the former period, and thus pressing back the air. A distinct cracked-pot sound has been described in several cases, but only when there was an opening in the pericardium. In the recumbent posture the extent of tympanitic resonance is greatest in front. When fluid is present, if the patient be slowly raised to the sitting posture and made to lean forwards, this area diminishes progressively, and the clear sound is replaced below by the dulness of fluid. Lateral changes of position will modify the relations of gas and fluid in a similar way, and thus very rapid and striking changes in the situation and relative limits of the respective percussion sounds are produced. Metallic instruments have been used to bring out the peculiar characters of the percussion sound.

(v.) Auscultation signs are also very peculiar, and often remarkable for their loudness. They vary according to the relative amount of gas and fluid in the sac, and the consistence of the latter; but as a rule different sounds are audible. If there be but little fluid the heart-sounds are abnormally loud, and are accompanied with a clear metallic ring, compared to a chime. Should there happen to be an endocardial murmur or friction-sound, it will probably assume a similar quality. The agitation of fluid and air within the pericardial sac by the action of the heart, and also by deep inspiration, produces adventitious sounds of the most extraordinary kind. They are all of metallic ringing quality, and have been described in different cases as splashing, spluttering, guggling, gurgling, rattling, large crepitating, and churning. They have been likened to the sound of a water-wheel or mill-wheel (*bruit de roue hydraulique, bruit de moulin*); and in one case to the "shaking of shot in a shot-pouch." Occasionally metallic tinkling has been noticed, due to the dropping of fluid in the pericardial sac. From a case observed by Dr. Flint, in which recovery took place, it would appear that sounds of the character just described might be produced by the presence of air and blood in this sac. In some instances the cardiac and adventitious sounds are so intense as to be heard, not only by the patient, interfering with sleep, but by those about him, or, it may be, even at a considerable distance off. Sometimes a splashing sound is brought out on succussion; or a bell-sound can be elicited by percussion with coins. It is affirmed that the signs of pneumopericardium have followed those of pericarditis, namely, friction-sound and evidences of effusion, when it is supposed to have resulted from decomposition of fluid.

Diagnosis.—If the physical signs just indicated were always pronounced, the diagnosis of pneumopericardium and its accompaniments would be quite easy. Otherwise it would present much difficulty, or mig be impossible. No reliance can be placed on symptoms. The only con tions with which it could possibly be confounded are a large cavity in t lung, in the vicinity of the pericardium ; a localised pneumothorax ; a greatly distended stomach. Due consideration of the general circu stances of each case, and of the clinical history and phenomena, shou obviate any such mistake.

Prognosis.—This is obviously very grave, and the termination almost always fatal, especially as the pneumopericardium is usually complication of some grave disease. A few cases of supposed recove have been reported, but these have been chiefly of traumatic origin.

Treatment.—But little can be said under this head. The patie must be kept as quiet as possible, and in the position most comfortab to him. Stimulants, sedatives, or cardiac agents should be administer as circumstances require, but each case will dictate its own methods. T question of operation naturally presents itself, and in suitable cases might be desirable to let out some of the gas by means of a fine troch the patient being in the recumbent posture ; or to open up the pericardiu freely, especially if it contain inflammatory or other products of a lo type. This matter must be regarded and dealt with entirely from surgical point of view.

VII. NEW GROWTHS AND PARASITES

In order to complete the account of diseases of the pericardium so reference must be made to the **morbid growths** which may affect it. the same time it is difficult to say anything of clinical importance, a a few general remarks must suffice. The reader may also be referred the article "Diseases of the Mediastinum," in the sixth volume.

Tubercle is by far the most frequent morbid growth met with in t pericardium, and perhaps in its minor degrees it is more common than usually supposed. It is only in exceptional cases, however, that t membrane presents gray granulations in general acute miliary tuberculos In the large majority of instances tubercle of the pericardium is chron and secondary to tuberculous disease elsewhere, especially of the lung from which it spreads directly. It may, however, follow disease of t bronchial or mesenteric glands. A simple pericarditis appears to more common than tuberculous, even in cases of pronounced phthisis ; a chronic inflammatory products in the pericardium may possibly becom infected with tubercle. Dr. Habershon records an interesting case general tuberculosis affecting unusual structures, where there was exte sive tuberculous pericarditis. In a case of phthisis which came und my observation, changes due to chronic pericarditis were well marke but careful examination failed to detect any tubercles or tubercle bacill In some instances gray and caseating tubercles are scattered over t serous coat, or in the midst of inflammatory products or bands adhesion.

Carcinoma of the pericardium is extremely rare, and always secondary. The sac is nearly always involved by extension from neighbouring structures. A growth in the heart walls may project into the pericardium; but most frequently this structure is implicated during the progress of a mediastinal tumour, or one starting from the œsophagus. Exceptional cases are those in which cancerous nodules appear on the serous surface, associated with a similar condition of other serous membranes, these being secondary to cancer elsewhere. When the growth results from extension, the parietal portion of the pericardium usually presents a diffuse infiltration, but occasionally a nodular mass projects into the sac.

A case of malignant sarcoma of the pericardium, believed to be primary and independent, has been described by Sir W. Broadbent (14*a*).

Hydatids of the pericardium are so rare that out of 1897 cases collected by the late Dr. Davies Thomas of Adelaide, in only two was this structure affected. Moreover, in no instance had a hydatid cyst in the cardiac walls ruptured into the pericardial sac, probably because of adhesions between the two surfaces. This writer mentions one case, however, in which a cyst situated between the liver and the diaphragm ruptured into the pericardium.

The effect of any new growth in connection with the pericardium would probably be to set up inflammatory changes. These have already been fully discussed, and it will suffice to state here that they are very seldom acute; they may be subacute, but by far most commonly are chronic in their development and results, constituting the ordinary forms of *tuberculous* and *carcinomatous pericarditis*. The combinations in these chronic cases of adhesions, pericardial thickening, and localised collections of fluid, along with the morbid growths, may be very complicated. The effusion is commonly hæmorrhagic; but in malignant cases it may be purulent or ichorous, and possibly also in those of a tuberculous nature.

Clinically new growths in the pericardium could only be suspected or recognised by the appearance of symptoms and physical signs of pericarditis, especially chronic, in such cases as tuberculosis or old phthisis, or an intrathoracic tumour. It certainly is desirable to watch the pericardium in cases of chronic phthisis, though, as already stated, the changes which may then arise are by no means always tuberculous. It is very likely that tubercle or cancer may produce a friction-sound, and this has been definitely asserted; but no definite diagnosis could be founded on this sign. The implication of the pericardium in these growths, in cases where the primary seat of mischief is away from the chest, could only be made out by the occurrence of pericarditis and its consequences, which would draw attention to this part.

Treatment is entirely symptomatic and constitutional, and no definite rules can be laid down. Operative interference might be indicated for the removal of fluid to give temporary relief, but nothing can

be done for the growths themselves. Obviously when the pericardi
becomes involved in malignant disease the end cannot be far off.

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FUNCTIONAL DISORDERS OF THE HEART

To the purist the vulgar distinction between functional and structural disease is a false one. We are assured that in every change of function a change of structure is implied; indeed, that structure and function are one, and to use them severally is to see the same thing in different aspects. It is not so much that the materialist and the idealist have lain down together, as that the idealist has swallowed the materialist. Yet, granting all this, we remember that as it is convenient to detach the study of physiology more or less from that of anatomy, so it is with nosology when we analyse symptoms apart from morbid anatomy; although we shall not forget that knowledge thus obtained must be integrated by bringing the two studies together from time to time.

Furthermore, we shall not be discouraged from using the term functional disease in a still narrower and more artificial sense,—in the sense of a perturbation of a more or less contingent kind, of a contingency sufficient to rock but not to upset the moving equilibrium (11). Every beat of the normal heart is a disturbance of equilibrium, and we do not forget that, in any system, cessation of all disturbance is the peace of death; on the other hand, disturbance beyond the resistance of the equilibrium is disease or death also. Between the death of apathy, as of the old man who falls asleep, and the death of defeat, as of the man who succumbs in his prime to a clot in the pulmonary artery, there may be two periods,—the period of health and the period of transitory discord. In health the disturbances are rhythmic, harmonious, controlled; in functional disease they are arrhythmic, uncontrolled. In functional disease the going system halts or staggers, but not beyond recovery; the humming-top swerves under a puff of wind, or reels as it travels over a grain of mustard seed; but the deflection is counteracted, and is presently resolved. Such temporary eccentricities are common to the heart with other organs, but are more conspicuous in the heart, because its workings are nearer to our consciousness, and lie, moreover, in the track of emotional gales and typhoons. Is there a man so stoutly knit, whose inhibitory nerves are so powerful and alert, that in passion or "twixt doubtful fear and feeble hope" he has never felt his heart climb into his throat? Thus it is that functional disorders of the heart are familiar to us all, and occupy our thoughts the more, as the heart tells us where the centre of life is, and where we cannot afford to have things go wror But it may be objected, and in a very important sense truly objected, th these are but matters of degree—that persistent functional disease ends structural disease. With this inquiry we shall deal at length ; meanwh I would say that this is not necessarily so. While, on the one hand, warn the student not to overlook the stealthy inroads of structur disease, of "functional" disorders, which are the first signs of t invasion of structural disease — such as retardation of the heart, f example, on the other hand, we shall not put them in the same reckonir with the functional disorders which are not of this kind—such, for exampl as acceleration of the heart. Whether a purely functional disorder b damnable iteration can hammer disease, as it were, into a harassed organ hard to say; as yet we can only say that in many cases a lifetime of fun tional disorder of no little persistency is not long enough to bring th event about, and perhaps that such is the usual issue : on the oth hand, it seems no less certain that perennial depressing causes, exile bondage in an invisible Babylon, may induce degenerative changes the heart and blood-vessels, or in the kidneys, as I alleged in 187 and have had yet more reason since to believe. That tachycardi usually perhaps when severe, may wear out the heart is true ; yet scarcely think we can regard this truth in the light of our present arg ment, as such gradual inroads are rather of the nature of dilapidati than of mere disorder: moreover, in a particular case it may be ha to distinguish between a perturbation, such as a variation in rate, which an indication of incipient heart failure, and a perturbation of central eccentric nervous origin. Anxiety long continued seems to perve nutrition at its sources; perhaps to prevent healthy metabolism, and favour auto-intoxication with its damaging effects on kidney and hea Such influences, however, come rather under the head of the remoter caus of diseases of the myocardium than under that of functional disease of t heart; as, again, many of the conditions of functional heart disorders w be dealt with in the chapter on Neurasthenia. For our present purpo functional disease may be taken to include temporary irregularities of rat rhythm and tone, and even of force and volume; though these last rath pertain and are subordinate to other diseases—that is, to other sympto groups. While rate, rhythm, and tone make important parts of mar maladies, yet their errors are often themselves the leading morbid featur and appear to the patient, and often indeed to his medical adviser lik wise, to stand almost alone. For instance, if in the irritative stage meningitis we mark a slow pulse, we do not group this phenomenon wi functional disease of the heart, however logically we might do so; th to class it would be to darken our conceptions, to introduce false conn tations. So again the quick pulse of a later stage of meningitis, or th of pneumonia, will in like manner be classed, not with functional diseas of the heart, but with the phenomena of fever. For our present concer is with clinical medicine, not with the broader views of gener pathology.

A more difficult problem of nosology is to decide where we are to place the quick pulse, say, of larval Graves' disease; if both goitre and exophthalmos be absent, as often they are, are we in the presence of an obstinate case of functional disease of the heart? Again, I think that to speak thus would be an abuse of terms; if we suppose that on due analysis this pulse has affinities with the symptom group which we call Graves' disease, we must not put the pulse characters in an independent category; we shall regard them as a part of that other group. Let it not be said that this discussion is otiose; for if the argument be well founded we shall no longer allow ourselves to call any quick pulse "tachycardia," nor any slow one "bradycardia." Tachycardia, for instance, appears to be a definite and primary functional disease of the heart; the affection has characters of its own: whether bradycardia is such a substantive malady is less certain; this question we shall discuss presently. If it is not, the specific name should be given up, as one without a consistent signification.

We cannot consider the heart apart from its nervous connections; like a well-handled pair of horses, its good going depends as much on the man on the box as on the muscles in action. Although the heart muscle has an independent and inherent rhythm of its own, this rhythm goes astray if the organ be severed from its nervous governance. The inherent rhythm may suffice for less complex organisations, but it will not do for a mammal. In the higher animals, for instance, the contraction of the left ventricle, although it is always a maximum effort, does not at every beat supply the whole arterial tree. That at a very low pressure, all the arteries being expanded, it might do so is possible; some of the strange perturbations of women attended with heat and flushing may thus come about; but probably even in them the distribution is more or less partial. In health, at any rate, the output is turned now here, now there, as—if I may be permitted so unsavoury a simile—in a sewage farm the fertilising streams are diverted by locks in this way or that. The lock-keepers belong to the nervous parts of the cardiac machinery. In study the active brain, after a meal the stomach, demand their alternative streams; by means of the nervous system an anæmic area calls for more blood, satiated areas for less; and by means of the vagus nerves the heart itself is protected from too great an importunity. If in an anæmic girl the heart beat too fast, we shall not call that a functional disease, which is an attempt on the part of the heart to respond to the cries from anæmic areas all over the body; though in many such cases as this we do for the moment, and provisionally, apply such a name to mark a region of our own ignorance.

As we carry our explanations into such regions we gradually diminish our group of functional diseases of the heart. Let us consider the effect of certain poisons on the heart. In so far as these and their effects are known—as, for example, in the cases of coffee, tea, and tobacco,—we shall scarcely call their ill effects on the heart functional disease of this organ; we shall turn rather to the chapters on these drugs, and regard the cardiac perturbations subordinately as features o the symptom group or series of groups associated with the agent concerned. Now the heart is often set on edge by obscure causes which seem to u to be of the nature of poisons, of poisons generated, perhaps, in th body and circulating in the blood which irritate or depress the hear directly; or, perhaps, disturb it indirectly by some obscure interferenc with the blood-pressure: such a state of things is surmised to exist i the malady popularly known as "suppressed gout." But when we know all about "suppressed gout" and wherein it consists, we shall remove th cardiac phenomena from the chapter of functional diseases of the heart and put them in their own place as subordinate phenomena of the gouty group. All we know about "suppressed gout" at present is that it is no a mere dilution of articular gout; that, however related to the latter, i is a different disease rarely occurring in the same persons; or, if in th same, at different times of the life of the individual. Cardiac disturbances often appear, it is true, in articular gout also, and are described i treatises on this disease as "gouty"; but of "suppressed gout" high arteria pressure is characteristic, from articular gout high blood-pressure i commonly absent. How terrific and how various may be the effec of the poisons of certain of the infectious diseases upon the cardia mechanism is familiar to us all. In diphtheria the heart's action may b reduced "almost to extinction" (Powell), and of the effects of influenza i the same direction an excellent account is given by Dr. Sansom (11) Syphilis, again, is said to cause irregular heart, as a functional disorde apart from arterio-sclerosis. Of this I have no personal knowledge Such considerations as these seem to threaten the very existence o Functional Diseases of the Heart, save in the sense of a survey of th general behaviour of this organ under all sorts of maladies, not excludin its own structural diseases. Meanwhile, however, we must deal with th unrelated cardiac disorders in a somewhat miscellaneous way; and certai of them seem to have an individuality of their own. Before studyin functional diseases of the heart as groups of symptoms, we may profitabl consider the elements of the groups separately—such as tone, tension rate, rhythm, volume, and so forth.

Tone.—The old-fashioned word "tone" has fallen into disuse; th more is the pity. When I was a student we were asked how the puls might be for tone; now if a student be asked such a question he talk about "tension," although he does not clearly know what he means. T measure or even to estimate roughly the degrees of stretching of th coats of an artery is a very complex and usually an insoluble problem yet to these coats only can the word "tension" apply. The blood itsel cannot be tense in any but an abstruse mathematical sense, which n student of this subject has in his mind. If the radial artery contrac tightly on the blood within it, the pressure on each superficial unit o

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[1] A pair of interesting tracings is published by Dr. Waller on page 32 of his *Physiolog* (ed. 1897); the first of irregular and low pressure pulse under tobacco, the second a correction of the same pulse under digitalis.

internal surface is increased no doubt; but this is not tension, or at any rate not in any simple sense. Tension is that stress which tends to split the artery either longitudinally or transversely; and such stress is at more advantage when the vessel is relaxed. Tension and tone have, indeed, something like an inverse relation one to the other, as we see more readily, perhaps, in the ventricles of the heart. We may say, indeed, that one of the chief functions of tone is to resist the tension which calls it forth. How tension acts upon an artery is best seen in aortic regurgitation, in which malady the effects of tension seem at their highest. We have but to look at any long artery in an advanced case of this kind to see what tension, in the longitudinal direction, really is; the artery is not actually split transversely, perhaps, but it is lengthened enormously and thrown into curves. No doubt, under all circumstances, whether the radial artery be tight or slack, there is more or less tension of its coats; but it is most difficult to ascertain the degree of this, even roughly: yet such is the love of obscure diction, that, instead of endeavouring to express the facts in terms as comprehensible as possible, that factor which is at once the least appreciable and the least immediately important is chosen for description.

Without saying that any factors in this application of hydrostatics are easily estimated, we may assert that tone and blood-pressure are easier to measure approximately than the tension of the arterial coats. The finger can tell with some approach to accuracy whether the pressure be low, moderate, or excessive, though it is only by such instruments as the sphygmometers of Roy, Leonard Hill, or Oliver that the degree of it can be recorded. Tone, again, is easy to make some guess at, or even to formulate with sufficient accuracy for clinical purposes. Tone in a vessel is that which preserves its mean diameter, which preserves a certain proportion between the extremes of dilatation and recoil, and which has furthermore the somewhat different virtue of keeping the vessel well home upon its contents. Therefore when we speak of a pulse of good or ill tone we are not talking altogether of what we do not understand. We mean that the difference of pressures between the base of the pulse-wave and the apex is somewhere about 35 mm. Hg. And again, when we speak of high arterial blood-pressure we are talking of that which we can estimate with some correctness—namely, a mean pressure of about one-eighth of an atmosphere. These two conditions the skilled finger is able approximately to ascertain. But when we speak of the tensile stress on the walls of a vessel we are talking in the dark; other things being equal, the higher the blood-pressure the more the tensile stress, but until we have allowed for tone the net tensile stress, however considerable it may be, is inappreciable. Now, in functional disorders of the heart and arteries tone is often signally deficient. The aorta, structurally healthy, may nevertheless be seen beating diffusely in the episternal notch and in the epigastrium; the wall of the chest may thrill as the hand is laid over the heart; the sounds of the heart are carried far along the vibrating walls of the carotid; the abdominal aorta leaps like an aneurysm; nay, even the patient himself may complain of the bounding of slack arteries all over his body. In some such cases even a capillary pulse may be seen. To the finger the radial or other artery is ill-filled, and the sphygmographic curve shows that the due proportion between the expansion and the recoil of the vessel is no longer preserved; the lever falls almost to the abscissa before the dicrotic wave is formed. I have often seen a temporary extension of the area of cardiac dulness in such cases. This state of the circulation is perhaps never so primary and eminent as to amount to a functional disease of the arterial circulation, and as a derivative condition its importance is discussed under Chlorosis, Neurasthenia, and elsewhere. The mechanics of the subject will be dealt with hereafter by Dr. Leonard Hill.

Tone, Dr. Gaskell tells us, is innate in muscle, but it may be excited, raised, or reduced by nerves. Tone may vary under nervous governance, but it persists beyond all nerves. Some of Dr. Waller's experiments suggest that nerves like muscle may have their refractory periods, and the same character has been indicated by certain observers, for example by Richet at the Toronto meeting of the British Association in 1891.

Rate.—How widely the rate of the heart-beats may vary between its extremes is too familiar to need description. In one bed may lie a patient with a pulse of 30, in the next one whose pulse is 180; and even these are not the utmost extremes. Under bradycardia and tachycardia we shall discuss those phenomena more intimately. The most general factor in acceleration of the heart is loss of vagus control, for the vagus may be regarded as the escapement of the arterial train. Loss of vagus control may be relative or positive; the accelerator nerves may be abnormally stimulated, and thus may overbear even a normal vagus control; or the vagus may itself be more or less in abeyance, as after a dose of atropine which palsies its ends in the heart. Again, agents acting directly on the heart itself may either stimulate the vagus, and so slow the pulse, or may overbear its control and the pulse-rate may rise; variations in blood-pressure have these effects, an increase of pressure tending, as a rule, to the retardation of the heart, and a fall to acceleration of it. In functional heart disorders we are frequently met by problems of this kind, and sometimes they are very difficult to analyse; we may remember, however, that controls are a later development than the functions below them, and therefore tire sooner. Vaso-constrictor action never tires so long as the nutrition of these nerves goes on, and the vagi tire before the accelerators. Thus the accelerating nerves often fatigue the vagi and run away with the heart. This may be the explanation of rapid pulse in certain poisonings, infections, and the like; but we have also to remember that in fever blood-pressure often falls also, probably from some change in the viscosity of the fluid; and again that quasi-normal catabolic products may act directly on the heart, as we believe that fatigue products do. That states of the cardiac muscle itself are often directly concerned in its rate seems also probable from the clinical phenomena of "irritable heart," which can scarcely be due to fatigue products only. Conversely fatty degeneration of the heart is often betrayed by retardation of the pulse.

Once more; we have to deal not only with the nerves, but also with the cardiac centre in the bulb, a nervous factor which may conveniently be considered apart, as through its efferent fibres it is chiefly concerned in regulating response to the demands of the system. Not in the case of circulating poisons only, but also under the fluctuations of ordinary blood changes, the cardiac centre is constantly in exercise. In hæmorrhage or chlorosis, for instance, the call of extensive anæmic areas throughout the body,—the afflux, in this case, of impoverished blood to the cardiac centre,—excites the centre to quicken the heart. On the other hand, a rise of arterial blood-pressure stimulates the vagus roots in the bulb, and the pulse is slowed. The name tachycardia, as we shall see, is improperly applied in the sense of mere rate; it is the name of a particular disease. The name "embryocardia," which is creeping into clinical language, is pedantic if it means merely a very rapid heart, misleading if it suggests that the heart has undergone some reversion to a fœtal quality, or even that the organ is primarily failing. The heart goes "tic-tac" whenever its rate reaches a certain degree, and I may repeat that a quick heart is not in itself a sign of enfeeblement, but of extreme reflex excitation of the accelerantes, due probably in typhoid and the like to a diminution of the total volume of the blood, or to alterations of its density, though, no doubt, the effects of morbid or catabolic poisons often intensify the state. It must be remembered that a rapid rate does not necessarily mean an increase of total work done: on the contrary, although dilatation is no uncommon result, hypertrophy, in the absence of valvular disease, is rare.

Abnormal rates of the heart depend then on many factors, and the variation of any one of these will modify the action of the organ under observation.

Rhythm is not synonymous with rate, as is too often assumed. A few weeks ago I read a valuable physiological essay in which rhythm was used almost throughout in the sense of rate; such abuse of language leads to confusion of thought. Rhythm is not the rate but the proportion of motion. Strictly, force and volume are contained in the conception of rhythm; but custom and convenience have ordered that by rhythm we shall mean the numerical proportion of motion; that is, a true cardiac rhythm shall consist of the same number of beats in every unit of time. Here again, although we find that the vagus is chiefly concerned in the variations of rhythm, such variations being due for the most part to vagus interference, yet, as in the case of rate, we learn that the rhythm at a given moment is due to a composition of causes which are not always easy to analyse. For instance, clinical experience suggests to me that intermittence of the heart is often due to a direct effect on the cardiac muscle itself, or is a compound effect of direct influence on the heart and vagus together. Digitalis may be an instance of an agent acting in su a double fashion, and some morbid poisons, such as that of influenza, sec to have a like compound property. Intermittence, transient as it usual is, is no uncommon feature in the degenerate heart.

We divide disturbances of rhythm into "irregularity" and "int mittence," terms which speak for themselves. That these two abnormaliti may be and often are present together is familiar to every student. Ir gularity of rhythm is for the most part graver as a sign of disease th intermittence. Its signification in muscular and valvular disease of t organ in chorea, in cerebral disease, and so forth, will be discussed the several parts of this work which deal with such subjects. I ne scarcely say that there is an irregularity of the radial pulse and another the heart, and herein we see that irregularity is not only an alteration rate, but also of volume and force; the ventricle not only acts irregularly time, but also delivers variable quantities of blood with variable impuls the output is unequal. There may be, as in cerebral disease, for instanc an irregularity of time only, the volume and force remaining constant; b such a condition is rare, for if equal quantities of blood are not deliver from the several chambers in equal times, inequalities of distribution the chambers and of systolic output must accumulate. Strictly speakin no pulse is regular, as a time line at the foot of a sphygmographic traci will prove; if not otherwise influenced, the respiration at any rate distur the order, as does muscular effort, even the slightest, especially in nervo or otherwise unstable systems. To ascertain how far the effort a position of the upright attitude, or a slight muscular exertion, quicken t rate is a good test of vascular resistance; for Dr. Waller's electroton work brings out into more prominence the truth that increased capaci is associated with diminished susceptibility to contingent impressions, su as relatively slight changes of blood-pressure. At the same time, it seen that in some persons the pulse is habitually irregular in the clinical sens Sir Thomas Watson mentions such a case in a brother of his own; wheth the brother was a tobacco-smoker his distinguished kinsman does n record. In my own experience I have often met with an irregul pulse in smokers—never, I think, in the normal state. In acute disea irregularity generally means irregularity of output, and warns us of evi probably of dilatation of one or both ventricles.

Intermittence is often of grave augury, no doubt: in suspected cerebr disease it is an alarming sign; it is a grave sign in any acute diseas especially in the pulmonary attacks of the elderly; but in cardiac disea it is of less gravity than irregularity. It is common enough also dyspepsia, in suppressed gout, in smokers, and even in persons in who no flaw is to be found. I once found intermittence in two brothers wh came together to me for life insurance; both of them were very angr with me for refusing them, or rather for stating the facts which led their refusal by the company. Neither were smokers, or very moderatel so, nor were they large tea or coffee drinkers. They were vigorous youn men, their digestions were good and their teeth sound. The intermissio were occasional, on an average about one in thirty or forty. Perhaps no one passes through life without an occasional sense of cardiac intermission; and therewith is often found, though at much longer and more uncertain intervals, a flutter, felt rather in the epigastrium than about the heart. This flutter seems not always to be cardiac; there may be some alternative machinery for its production: sometimes it is certainly due to a series of rapid and irregular beats, but the disturbance is so quickly over, so hard to catch, that its precise causation is undetermined. This flutter, like the intermittence which is often associated with it, is of dyspeptic origin; and the best remedy for these discomforts, for they are little more, is to insist on slow mastication. They are very apt to arise in persons who bolt their food. It is incorrect to say that if such intermittence arise in advanced life it necessarily signifies incipient cardiac degeneration, for even in cases when the symptom has endured for two or three years in persons of sixty years and upwards, careful attention to the diet and a vigilant supervision of the use of coffee, tobacco, and the like, will spare them to die at a riper age of some other symptoms; on the other hand, even in much younger persons, intermittence may accompany vascular deterioration, cardiac strain, or valvular disease. Sometimes the intermittence is radial only; the heart beats regularly, but not always effectually. Sometimes the intermittence is rhythmic; it will occur every two, three, or four beats for a while; such an intermittence is often found in persons under the use of digitalis. As a functional disorder the form is insignificant, or no more significant than ordinary sporadic intermittence. To say that the "pulsus bigeminus," the "pulsus trigeminus," or the "pulsus alternans" is a sign of cardio-arterial degeneration, to assert that it is necessarily significant of grave cardio-arterial involution, is to ignore daily experience. If indeed it be associated with an abiding or persistently recurrent retardation of the pulse the prognosis is less hopeful, as it may be also when such coupled intermittences obstinately return in spite of treatment. I had written these lines when a pamphlet by von Noorden came into my hands, giving descriptions and sphygmograms of such pulses in hysterical cases (9). It is said that an intermitting action which does not reach the consciousness of the patient is of worse omen than that which attracts his attention. Many persons are alarmed by a perceptible intermittence, especially by the bounce which often follows it; perhaps it is this bounce or thump, rather than the intermittence, which gives rise to the well-known sensation. Certainly that the comparatively harmless intermittence is perceptible enough common experience tells us; and I have noticed that intermittences occurring in failing hearts are less obtrusive or indeed unfelt; whether the absence of the sensible bounce indicates a feeble heart in all instances is more than I can say myself or find in the records of other observers. Certainly in the intermittences of acute disease, as of senile broncho-pneumonia, the missing beat is not perceived by the patient. The mechanism of intermittence is not quite understood; it is probable that a beat occurs, but is abortive, and that the bounce is a leap of the heart against the low pressure of the unfill arteries. The sign is more ominous when associated with irregularity.

Palpitation.—This disorder is even more common than intermitten in greater or less degree it lies within the experience of every one. It more common in women than in men; and in the former is oft a very distressing and persisting torment. Under the alarm of severe attack of palpitation, with its no less painful sense of choki even long and trying experience is scarcely enough to steel the pati against the dread of its return. Indeed, as the gale in which the he is caught often arises from the quarter of the nervous system t apprehensions are disordered as soon as the heart itself, or even bef it. A sensitive woman, physically courageous perhaps, yet one w starts at every sudden sound, may well be appalled by the fear of he disease and of sudden death. Attacks of palpitation often pounce up the sufferer in a moment—even in a quiet moment—and, it may without apparent cause. It is no unusual thing for an attack to set with nightmare during sleep. Either thus, or more gradually, the he begins to throb tumultuously, and its function is often beset in all t directions in which we have been regarding it; it becomes irregul intermittent, variable in force, volume, and rate, though always rap until the vagus control is regained either by the lapse of time or by so reflex stimulant such as smelling-salts, or a cordial; or again by so pain or conflicting impression. The attack may subside gradually, or may cease suddenly with a shock, as if rending the patient bef quitting her body. Such a finish is usually seen also in tachycardia, a may be due to the same causes as the throb of an intermittence. T patient instinctively presses her hand upon the region of the he during palpitation; a kindly pressure seems to soothe the tumult. Un the hand the heart's beating, like the arterial pulse, is vibrati diffused, turbulent, and disorderly; now striving and violent, n tremulous and faint. The attack is followed by the calm of exhausti The history and circumstances of such seizures are generally enough serve us for interpretation; indeed, such storms are unusual in orga cardiac disease. Still, the static conditions of the heart are not often be appraised during the discordant and confused dynamics of su seizures. It is well, in the case of a new patient at any rate, to postpon final diagnosis till the ship is in calmer waters.

Murmurs are often present in the palpitation of functional disea they may be heard at apex or base, and at any part of the card revolution. A systolic murmur at the apex is the most frequent of the The causation of these transient murmurs is unknown; some may "anæmic"; some may be due to inordinate action of the papilla muscles; some again may be "pulmonary" (Potain). Until the patie is tranquil, and the physician at liberty to map out the heart and listen to its sounds without embarrassment, no final opinion should given. In a functional case the murmur will probably then have ceas and dilatation, if any, will be reduced; although resonance of the seco sound at the apex and the sharp knocking quality of the systole will probably mark the case as neurotic.

Of these murmurs Dr. Sansom says (13) that a systolic murmur, arising independently of structural disease, seldom attains its maximum audibility at the exact apex, but slightly to the right and left of it. It is usually soft, and does not replace the first named. Again, it does not occupy the whole, but the middle of the systole ("it is meso-systolic"). It is much influenced by respiration; it is intensified both during expiration and inspiration (especially the latter), but it often becomes inaudible at the end of an expiration. I may add that to me the quality is often that of the apex first sound in those cases of systolic murmur generated at the base in which the murmur is scarcely audible, as such, at the apex, yet where the first sound is blurred by it. For Potain's elaborate and almost too ingenious doctrines concerning the pulmonary origin of such murmurs—anæmic and the rest—the reader is referred to his well-known article in the *Clinique de la Charité*, 1894.

The immediate *prognosis* can rarely need much direction. Generally speaking, the *diagnosis* in such cases is too dark; a woman is told that she has got a "weak heart," and thus the confidence in herself which is essential to her cure is shaken. The palpitation of chlorosis I am accustomed to regard as the result of the combination of poverty of the blood in oxygen value with persistent mass, with no less a demand upon the heart, that is, in respect of output. The *treatment* during the attack consists in recumbency, warmth to the legs and feet, and such stimulants to the abated vagus nerves as ether, ammonia, valerian, smelling-salts, and hot applications to the cardiac region; remedies which are rather to be recommended than alcohol. Belladonna also is better avoided, and digitalis, if an occasional ally, is not to be trusted. In acute attacks these measures will suffice; but in some cases the palpitation does not take the form of isolated attacks, but, though less violent, is either persistent or chronically recurrent. In these cases treatment, if addressed still to the vagi, may well be addressed also to the accelerators, especially if the pupils be dilated and the face flushed, and thereby excitement subdued. As palpitation, if consisting partly in defect of central control, is nearly always set up by some eccentric cause, rules for general management, such as regulation of the bowels and other secretions, attention to piles, uterine disorders, overwork; temperance in food and avoidance of alcohol; moderate exercise, cold baths, and regular hours of sleep, will be found in the articles on hysteria and other neuroses. At times such sedatives as aconite and the bromide of soda, ammonia, or camphor may be needed. Aconite has served me well in many such cases, and its use, cautious as it must be, may yet be more than occasional. With palpitation run other symptoms, such as precordial pain, panting, globus, vertigo, and perhaps even syncope—though I have never seen it under ordinary circumstances. During the attack the urine is scanty, but it is generally profuse after it, as in megrim and other neuroses accompanied by fluctuations of blood-pressure. Such symptoms receive full attention in other parts of this work. The causes of palpitatioı also are dealt with elsewhere. I will but remark that sudden vaso-motc changes either in the direction of constriction or relaxation, are commo incidents in palpitation, and perhaps common causes of it. When w remember that, in the bulb, the cardio-inhibitory, the vaso-motor, th respiratory and the gastric centres abut upon each other we shall feel n surprise that the functions related to all these centres should ofte influence each other or be influenced together. The expulsion of a worı has sometimes proved to be the cure of troublesome palpitation. Palpit: tion coming on for the first time in later life is a matter for anxiety, bı may be gouty or dyspeptic (bad teeth).

False palpitation.—It is not uncommon for patients, especially fc highly neurotic or neurasthenic patients, to complain of palpitatio although on examination little or nothing of it is perceptible, or th heart may be accelerated by some five or ten beats at most; yet to judg by the bearing of the patient the distress is acute. Such patient will probably complain of other hyperæsthesias, and of pains in othe regions, such as the head and back. In the cardiac region the patier complains of tightness and oppression—"precordial anxiety"—of urge heaving, or bursting of the heart, or of cramp in the part, in which the fear to die. Or the pains may be boring or cutting: the husband of suc a sufferer, in writing to me, tore out from his Bradshaw the advertisemer of a corset-maker, and drawing a dagger with its point entering the le submammary region, enclosed the picture as a graphic representation (his wife's agony. As many of these patients suffer from air hunger an pains in the chest and arms the cases melt into the class of "pseudo-angin pectoris." The attacks may recur many times a day, and are not difficu to appraise in the broad sense as neurotic: the story of the case rarel leaves much doubt of this interpretation. The blood-pressure rise during the attacks and rapidly falls as it passes off. But in my opinio the vaso-motor phenomena are not causes, but consequences—are of refle origin and secondary to the neuralgia or distress. As auscultation an other means of investigation reveal no change or but little, the intimat nature of these phenomena is not easy to ascertain. In ordinary palp tation, as the pulse rises, perhaps to 120, the pressure falls and the fac flushes; or the patient turns pale and the pressure rises; but neither c these events is seen in the false palpitation. Until a better hypothesi is suggested, we may suppose that there is some morbid susceptibility t the impact of ordinary stroke of the heart. There would seem also to be like hyperæsthesia in the vessels, as rushings in the arteries, whizzings i the head, and other "determinations of blood" are complained of, sens: tions perhaps due to slackness of the arterial walls.

Weak heart is used in two senses; as a heart of lax or even failin fibre, and as a heart subject to certain kinds of transient disturbanc Of the former we have not here to speak; the second is as follows:- The patient usually, but not always, a neurotic woman, tells us th heart ceases to beat; in the severer cases the patient is convinced of this and fears that each attack in turn may be fatal. Sometimes as the attack comes on the face turns gray, and the lips blench; in other cases illness is betrayed rather by the expression of apprehension and distress in the face than by any signs of organic disease. The hand is pressed to the region of the heart where pain may be felt; but often it is not so much a pain or a throb as a sinking, and the sinking is not at the heart only, but a general "lypothymia." She may also complain of pins and needles, of turning cold, and other evidences of irregular blood distribution. After a time the distress passes off and the patient recovers with that sense of extraordinary exhaustion which is so well marked in functional affections of the heart. The pulse during the attack is not very characteristic; it is certainly weaker, it falls more or less in pressure; and therewith it increases in rate a little—say 100 to 110; but it is not the pulse of syncope, nor indeed do these patients faint away; they gasp and return to life with a sigh or two of relief. Speaking generally, there is no danger in the attacks except that which lies in the habit of taking drams to cure or prevent them. Mrs. Gamp's prescription of two drops of brandy on a lump of sugar is too well known to these patients and their friends; the medicine is at hand and is assiduously administered, with the rubbings of the extremities, the hot bottles, and the like, which are grateful to these patients. And no doubt for the moment the alcohol is helpful; it pulls the heart together, or imparts something or other which may be mere Dutch courage, or something more mechanical; probably its chief effect is to dilate the arteries of the surface, and thus perhaps to divert the blood from the splanchnic areas into the arteries of the skin and limbs which were certainly for the time anæmic. It is not apparent, however, that constriction of the arteries is primarily at fault — a dilatation of the splanchnic areas may be. Almost as I write these lines I saw with Dr. Henry Head a very curious case of functional cardiac instability. A gentleman, aged thirty-three, apparently healthy in all other respects, but of nervous temperament, complained to us of breathlessness on ascents. No anginal or other pain. At nights he awakes with a sense of faintness or impending death. His pulse, while he is standing, is 130, and but little less on sitting down; but as he lies down flat the radial pulse instantly undergoes striking oscillations for two or three seconds, and then falls to a steady rate of 80. He has some reason to suspect that his nocturnal discomforts are due to a still slower rate of the circulation. Aspect healthy; no cyanosis. Does not smoke nor drink tea or coffee. The heart on examination proves to be free from any abnormal sign, unless it be that the apex beat is obscurely seen, and the impulse rather diffused. He has had attacks of the kind before, if not quite so severe, and has always been cured by going to sea. I thought that the effect of a well-adjusted abdominal pad might be tried. I have seen at least one other such case. As bearing on the conclusions of Dr. Leonard Hill and Dr. George Oliver, I may say that in one patient, who suffered much from heart sinking, as above described, to raise the arms was almost a certain means of producing an attack or a threatening of it: hence she assured me that she dared not raise her arms to kno at the outer door of a house. Until I read Hill's papers I thought tl was all moonshine; now I think it was not. Whatever be the underlyi conditions, the repeated taking of drams is very mischievous; it encourag the very oscillations in blood distribution which we ought to control the wet sheet, douche, regular exercise, massage, and such mean and after a few months or more of the dram-drinking the doctor is to that an unaccountable nausea and retching in the morning, and loo motions, either before breakfast or during the forenoon, are added to t tale of her symptoms. The next stage is that of pains and palsy in t legs and feet. Such is a common enough story. Some cordial the patients will have, perhaps ought to have; they are frightened out of th wits, and a stimulant seems their only help. Well, then, let us prescri ether, valerian, ammonia, or peppermint for the moment; and as t immediate anxiety passes away, attention to the general therapeutic nee of the case will, in a broader and more wholesome sense, ere long remo the need for dramming at all. It cannot be too strongly urged upon the patients that temperance, even to the point of total abstinence fr alcohol, is paramount in the treatment of neurotic cases: it is even more important condition in them than in the gouty. Cardiac neuro are nearly always part of general neurosis; in all its phases neuro means lack of inhibition, relative or positive; generally relative. I George Oliver's comparisons of the range of radial volume in the healt and in the unstable respectively are full of instruction in this respe It is possible that in some of such cases there may be a lack of suprarer incretion and a corresponding loss of arterio-vascular tone. But this is dark matter; I have even found the radial blood-pressure rise at t outset of an attack and fall again as it passed off. In these phases high initial pressure the patient is flustered at first and sinks afterwar An increase of the muscular reflexes is often seen in these patients, as the following disorder:—

Passing by gradations into, or even confused with the above derang ment, is that of **cardiac asthenia,** which, in a recent pamphlet, Da Cos has distinguished from irritable heart (4). The author says that f long periods the action of the heart in these sufferers is feeble; a feeb ness to be distinguished from the weakness due to organic causes, a again from that of lithæmia, gout, tobacco, and the like.

The affection generally manifests itself in those persons whose nervo system has been strained by worry or overwork; whatever the warni signs, the full brunt of the disease is often sudden in its incidence. T patient is prostrate in bed; all attempts at sitting up cause swooning a vanishing pulse. The heart's action is feeble; the pulse is small and s and generally increased in frequency. Although without pain there is sense of uneasiness in the cardiac region. The bodily temperature, well as the warmth of the extremities, is lowered. The breathing is u affected—a point of distinction from organic disease. "I am out of hea rather than out of breath," was the reply of one of Da Costa's patient

Insomnia is not infrequent. The patient rallies but slowly; two months in bed may be his portion, and months more of ailment before he recovers; for the issue is as tedious as the onset may be brusque. In some few cases the rhythm of the heart is irregular. The disorder may occur in either sex, and at any time of life between childhood and old age. There is no percussion dulness, the impulse is feeble. The first sound is short, lacking in volume; the second sound is not accentuated. Hysterical symptoms are conspicuously absent. In "irritable heart" the patient can get about, the heart's action is more obviously disordered, the impulse is jerky and diffuse, the second sound is sharp and distinct. Tobacco heart might resemble that under discussion, but in my experience the tobacco heart is more prominently irregular, and is often "irritable." In distinction from organic disease are the disproportionate prostration, the absence of dyspnœa, and the freedom from any œdema of the shins or feet. An apex murmur may appear in the functional disease as in almost any kind of cardiac functional diseases. The prognosis is good.

The treatment recommended by Da Costa is as follows: At first rest in bed; then, as some ground is gained, carefully regulated shower-baths are to be given. The next stage may be massage, but often some time elapses before this means can be borne. Then Swedish exercises and gentle riding on horseback can be arranged by degrees. Nutritious feeding is of course essential, and, in Da Costa's opinion, a generous allowance of alcoholic stimulants is necessary also. Among drugs strychnine "stands pre-eminent." The dose need not exceed $\frac{1}{30}$ gr., but it must be given continuously. Arsenic is the next best drug; iron is not usually indicated, and the need of digitalis, if any, is but occasional. Nitroglycerine does no good. Bromides, valerian, or even opium may be required under special circumstances. If I may venture to guess at the pathology of these cases, it would seem that a dilatation of the vessels in the splanchnic area is the most probable explanation of them, and an artful compression of the abdomen might be found useful. The observed uselessness of the nitrites may support this view of the pathology. My patient, who could not knock at the door (p. 820), was perhaps one of this class, and I think I have seen many cases of the kind described. The state of the pupils might give us some useful indications in such cases; my impression is that they are either dilated, or at any rate contract slowly and imperfectly. Diminution of the mass of the blood, with correspondingly small output, may, as for example in the acute fevers, be a cause of such cardiac symptoms, however sound the organ itself. In cases such as these we have a truly "functional" disorder; the heart may be healthy, but its work is upset by circumstances.

Irritable heart.—Since the publication of Da Costa's and Myers' well-known papers this derangement has been too exclusively attributed to muscular over-exertion. It seems, however, that we must divide the subject of irritable heart into two classes: the irritable heart of young persons now to be described, a very curable disease, and the "Soldier's Heart," to be described under "Mechanical Strain," p. 851, which is too often sherry and half a box of mixed biscuits at luncheon; the manly absorption of grown-up and more than grown-up doses of tobacco; the black coffee and cognac of an evening after a large gobbled-up dinner; the hot arguments on the framework of the universe and the destiny of man protracted till two o'clock in the morning; the spasmodic bouts of study; the examination bogie; the conflict with untamed and rebellious passions, some wholesome, some not so wholesome; the violent games and the bear-fights; the ardent hopes and the bitter griefs—what elder is there who recalls all these things, and does not long to dash pell-mell into it all again and accept irritable heart into the bargain? There is but one step between the wise young man and the prig, and this a narrow one; still that is no priggish advice which would cut out of this gay, ardent and careless life some of its idler and less lovely follies, and complete the cure of irritable heart by better-regulated exercises,—not violent stress one day, and idleness not unmixed with dissipation on the next, but regular training which shall promote a uniform development, not only of lungs and heart, but also of all the parts of body and mind. Muscular exertion, then, is a cause of irritable heart when it is pursued in an irrational and unsystematic manner by a more or less nervous and dyspeptic young person whose lungs are not big enough to carry off the blood as quickly from the right heart as it is delivered there; and whose ethical and intellectual life is lived after the same fitful fashion.

The irritable heart described in recruits, especially those suddenly removed from civil into military life—clerks turned into soldiers (Da Costa, Herz, etc.), is a different disease; and Mr. Simson Snell of Sheffield, in a private letter to me, says that colliers are very liable to an acute irritability of the heart, due probably to severe bodily efforts in awkward positions and in bad air. In these persons transient dilatation not infrequently becomes permanent (*vide* p. 851).

The treatment of the slighter and common forms of the malady is then one of regulated habits, and the avoidance of such poisons as alcohol, tobacco, tea, and coffee, except in doses which prove to be harmless to the individual. Muscular exertion must be systematic or indeed prohibited for a while or severely restricted. Of specific means none is required; it is better to avoid digitalis and the like, unless the symptoms be unusually vexatious, when small doses with a little bromide may be used economically. These patients are often a little shy and sombre in spirit; change of scene, pleasant society of both sexes, and frank and kindly advice on sexual matters, are a part of the services which a sympathetic physician may render to young men; for while we may have a kindly smile for their heroics, we must remember, nevertheless, that they are often acutely miserable. Some excellent remarks on this subject by Sir William Broadbent are reported in the *Lancet*.

The neurotic element in organic disease of the heart.—We are too much disposed to think that death from organic disease of the heart is the direct result of its utter demolition; that the crippled organ stumbles along until it can do more, and staggering under an intolerable burden

sinks to its rest. We are too ready to assume that the diseased hea
fails by means of its sheer mechanical inability. This may perha
be the case here and there. Dr. Solomon Smith has on more than o
occasion reminded us, however, that in many instances, at any rate, th
is not the course of events. To put Dr. Smith's view of the matt
summarily, he would have us see that the heart in advanced disease ma
fall, or stagger, under the intrusion of some neurotic accident, of som
nervous perturbation, whether of reflex or inherent origin. The harmon
between the reflex stimuli from the different segments of the hea
may and frequently does become deranged; and it is not surprising th
irregularity should result. Again, derangements of the stomach
bowels, torpor of the liver, pulmonary spasm, cerebral or bulbar inte
ference, the absorption of toxic products, and so forth, are potent
depress or disturb the heart's action far beyond its mere mechanical di
advantage. Thus it is that in most cases great oscillations occur: at o
time the patient is pretty well, at another he is at death's door; yet aga
he comes round, and this not necessarily as a result of treatment, or if
treatment, of such a remedy as an injection of morphine, which may
adjust or permit the readjustment of the harmony of internal cardi
stimuli; or may block some reflex arc with its superadded neurosis. Aga
the vomit of a little sour mucus or the discharge of an offensive stool ma
set matters right, even in a few minutes. It was with this conceptio
in my mind that in 1869 I recommended the subcutaneous injection
morphia in heart disease; not only does it, in appropriate cases, cut sho
a neurosal paroxysm of dyspnœa or restlessness, or restore the order
rhythm, and thus pacify the organ rocking under the tumult of its u
balanced parts, but it may prevent the heart from being "tripped up b
the intrusion of a neurosis," as Dr. Smith puts it. The complex rhyth
of the several parts of the heart and its allied vessels is but too easily broke
in upon at one or more points. The importance of these considerations i
respect of treatment is obvious. "Our choice of remedies lies no longe
only among cardiac stimulants or depressants, arteriole constrictors
dilators; a whole range of remedies is opened to us which, although wit
out direct action on the heart, relieve heart trouble all the same by remo
ing the starting-points of nerve derangements." I may add that not onl
are new remedial means thus opened out, but in these words we have th
explanation of the value of many remedies which, in a more or less em
pirical fashion, have long been familiar to us.

TACHYCARDIA.—The names tachycardia and bradycardia are ofte
used merely to signify rapid heart and slow heart respectively; such use
have not even the accuracy of pedantry. Dr. Herringham, indeed, think
that tachycardia is a "symptom rather than a disease," but in thus writin
he scarcely does his own monograph justice. If any rapid pulse, rangin
let us say, over 130, is to be decorated with this fine name there is a
end to clinical nomenclature. Dr. Watson Williams implies that tachy
cardia is a disease, but he prefixes the qualifying epithet "paroxysmal,

which from his point of view is superfluous. Dr. Herringham, in refusing to go beyond the bare etymology—the "prairie value"—of the name, argues, truly enough, that "no real distinction can be drawn between the cases with and those without . . . organic lesions." But is not this to deny also the validity of the names asthma, epilepsy, chorea? The author is right in warning us against the danger of "erecting a separate type" in such a case as this, a truth which I have endeavoured to emphasise in the introduction to this work; but, as I have there said, I do not think that the existence of mixed or transition cases forbids us the precise use of types. For what do we mean by "a disease"? Surely no more than the recurrence of symptoms in fairly uniform groups. A disease has no more "real" existence than has a constellation; stars, like symptoms, have a way of grouping themselves about centres of relative density; to such groups we give names, and no one should pretend that any disease has more than this relative or, if the reader please, this "subjective" existence. A type is an abstraction, an ideal pattern constructed from an infinite number of cases; and the moment we contemplate a particular case we leave type for embodiment: no two cases are identical, and no case corresponds in all respects with the type. Like Dr. Herringham, I am not fond of teaching by types, but they have their use in summarising and classifying our observations; and if we remember that they have no more claim to "reality" than this, we may use these conceptions without much harm. If, whenever we talk of "tachycardia," for example, the mind is to range over an indefinite scattering of cases in which the pulse is excessively quick, we shall waste a great deal of time in discussion and a great deal of space in books.

To what symptom group, then, do we apply the name tachycardia? Not to any case of quick heart, but to an enormous quickening of the pulses of a heart not necessarily the seat of static disease; a quickening which attacks the patient suddenly; which does not persist indefinitely, but for a variable space, rounded off by an equally sudden reversion to the normal state less certain phenomena of exhaustion. Heart disease, in the static sense, may coincide with tachycardia, it is true; mitral stenosis may coexist with chorea, nay, may even favour the occurrence or intensify the peculiar symptoms of chorea; but that surely ought not to deprive us of the name chorea, nor justify us in including under this name, as too often we do, any twitchings or gestures whatever which look at all like chorea.

Careful clinical observation and no less careful verification after death (so far as this has gone) indicate, at present, that attacks of rapid heart coming on suddenly, departing suddenly, and attended with certain other symptoms, objective and subjective, are consistent if not always coincident with a heart apparently sound; that coarse heart lesion is therefore not a necessary antecedent, or, in other words, is not a cause of this malady. Hypertrophy is not usual in rapid pulse, of any origin, as such; for increase of rate generally means diminution of output per beat, and probably per second also. If output per beat and per second is increased the rise in rate can be but moderate (Stewart). The records of necropsy in tachy-

cardia are few, no doubt, but we can only go upon such evidence as
have; and the sudden subsidence of these attacks without leaving behi
them any evidence of disease of the heart supports the interpretation
the scanty pathological material. The interpretation is that tachycard
is a fairly uniform symptom group; and, as one of its eminent characte
is its paroxysmal occurrence, the addition of this qualification to t
name is superfluous.

The attack is as follows:—As I describe it I have in my eye t
cases now under my care. The first is in a woman, passing (at the time
now write) through the menopause without any peculiar derangement, w
since her adolescence has been liable to seizures of tachycardia. She is
well-nourished person, and is now getting stout. Her anxious and fidge
temperament may indicate the neurotic bent, or may be the consequen
of her distressing malady; but the family history is without appare
bearing on the case. Her own life, though broken into more than once
calamity, has, on the whole, been one of prosperity; moreover, her ailme
dates from adolescence, years before these heavier trials had afflicted h
She is happily married, but has had no children. The attacks, which ha
preserved the same characters from her adolescence, are as follows:—S
turns a little shivery and pale, at times even ashy; and a peculiar lassitu
and restlessness possess her; the extremities are cold, and these and oth
parts are "numb." She soon becomes aware of a tightness, tremor, a
oppression rather than of a beating about the heart; the tightness m
amount to actual pain, and may dart here or there. The pulse is n
beating at the rate of 160 to 200 a minute (the reckonings of the pul
have not been systematic; and often the only record is that the pul
could only be counted at the heart). After the attack has continued f
a day or two I find that the area of cardiac dulness has extend
towards mid-sternum, or even beyond it; the sounds are tick-tack, but
added sounds are to be detected. As the attack goes on she becomes ve
fretful and wretched, but the oppression and tightness and other sig
suggestive of peripheral arterial contraction pass off. The urine in mo
cases is scanty; at first, perhaps, from contraction of the renal arteriol
later from low arterial pressure; but in her and in another of my cas
nervous polyuria attends the attacks throughout. It seems certai
from the change in the volume of the heart, that the residual blo
in the ventricle is large and the output correspondingly small. T
relief of oppression does not signify that the tension of the ventricul
walls and aorta is diminished, but that the sensibility of the heart
blunted. In severe attacks she is more or less aphasic, with the aphas
of exhaustion—a phenomenon not uncommon in megrim and in perso
spent by fatigue. Such an interval of aphasia is described by Tynd
in his own person after a dangerous and exhausting scramble among t
rocks above the Grimsel. The most complex of muscular co-ordinatio
give out early, as we might expect; but, as in many cases of nervous pr
tration, she has often a special sense of weakness or palsy in the left ar

The duration of the attacks is very variable. In some patients an atta may cease after a few hours or a few minutes; or again it may continue for three, four, or five days. It is said to have lasted in some cases as long as ten or eleven days; or indeed for weeks at a time, but suspicions of a wrong diagnosis present themselves on the consideration of such records. Perhaps the longest attacks of those carefully recorded were in a case recorded by Bouveret, in which they endured for thirteen days. In my second patient the attacks would return in groups, giving thus an impression of a longer paroxysm than was strictly the case. He might have a series of four or five attacks, and then none for a year or more. During the one or more nights of an attack the patient may be almost sleepless; but during sleep the tachycardia pursues the same course. Sometimes during these nights the female patient described above is a little delirious. The beating of the heart is regular in all cases unless the ventricle be dilated, when every pulse may not reach the wrist. In a case recorded by Dr. Bristowe the pulse number reached 308 a minute.

The cessation of the attack is always brusque, generally sudden; it may end in a few slow hard beats, or in one violent rebound, followed, says my second patient, by "a sort of swim." The trial is now over; exhausted as the sufferer may be, there is freedom—till next time. The urine in these two cases has never contained sugar, albumin, or any substantial excess of urates or phosphates. Attacks cannot be traced as a rule to any cause or to any season; they may come on at a moment of rest; often they begin or end during sleep. My second patient is an epileptic tailor, with a good family history. In him over-exertion often brings on an attack; but an attack thus produced can always be arrested by holding his breath in inspiration and then stooping tightly down with his belly on his thighs. Spontaneous attacks cannot be thus cut short, though once (the third of four attempts) he stopped one in my presence for a few seconds; the pulse fell suddenly from 166 to 80. As he rose up the rate as quickly returned.

Such is the ordinary course of a well-marked case of tachycardia, though cases of greater and of less severity occur. In one of my present patients there is no abnormality of the heart save the usual short, sharp action of neurosis; in the other there is a permanent apex systolic murmur, but no dilatation; in worse cases irremediable dilatation of the heart is brought on. During the severer attacks œdema of the lungs may accentuate the signs of dilatation, and later the feet may become œdematous, and albumin may appear in the urine. A repetition of such incidents renders the heart less and less able to recover its normal tone, and the symptoms of dilapidation set in which need no description in this place. Death may be by asystole, or by syncope; probably nearly always by syncope.

I may add to the story of my lady patient, that her first attack cut short a prolonged and severe skipping effort, when she had reached a high tale of skips. Ever since she has been subject to attacks, but they are not so severe as formerly. They seem to come on capriciously, she thinks more in spring and autumn than at other seasons. Sometimes they have been determined by a shock, physical or emotional, as once

when she made a false step in the street and "jarred" her foot; ar
once again when a drunken man seemed disposed to accost he
Dyspepsia may seem to call forth an attack, but, in both these cases, by f
the majority "come on of themselves." Her pulse generally runs abo
200; the highest that has been noted accurately was 280. The attacl
go off somewhat variously: either "hardly," that is, more gradually wi
a peculiar sense of agony, when she used to think she must die;
suddenly with a thump or two. The attacks may last for a few second
a few minutes, or a few days; some attacks have lasted as much
ten days, but this duration has been unusual. She feels conscio
enough of the beating; it is like a rapid tapping or vibration: when sl
was younger it would shake the bed and even the room. In latter yea
she can bear them better, no doubt they are milder; she can even re
during the attack. Formerly she was prostrate throughout the course
them, and long after them; indeed, they are all most exhausting. H
family history is very good; her parents are hale octogenarians. N
notable nervous disease has been heard of in the family. I described h
as a nervous and fidgety person, but she assures me that the tachycardi
attacks have been the cause of this disposition by the injury they ha
wrought upon her nervous system. This is probably true, as she
of sturdy build and well nourished. She has no permanent signs
cardiac failure. In the epileptic case the fits came on at æt. 42, the hea
attack at 14. The two maladies move quite independently of each othe

Morbid anatomy.—I have said that the evidence of necropsies is
yet scanty, though the two or three careful examinations on record agr
in indicating that, these evidences of cardiac decadence apart, no consta
changes are found. Examination of the vagi, of the sympathetic nerve
and of the intra-cardiac ganglia have been negative, except for seconda
changes such as the degeneration of muscle and ganglia in common.
then the evidence of the stethoscope is also negative, and as for ma
years the patients recover their ordinary health between the attacks, w
must regard tachycardia for the present as a functional disease. If t
ultimate prognosis be doubtful, if in a certain number of cases the eve
be death, the immediate prognosis, in the earlier years at any rate,
hopeful. Indeed, Dr. Watson Williams records a well-marked case in
patient aged—at the time of his writing—eighty years. The patient
have referred to is about forty-eight. In cases which, after the lapse
years, have proved fatal the necropsy may reveal, as in a case of Fraentzel
fibroid degeneration of the walls of the heart and dilatation of its caviti
in all directions. Broadly speaking, then, in tachycardia no primary
constant morbid lesion has been discovered. Ultimately the disease oft
wears out the heart; but where or how it is engendered we know not.

Pathogeny.—With the best will in the world I cannot follow t
example of recent writers in discussing the "theory" or "theories"
tachycardia. No theory exists. Certain surmises, such as no compete
physiologist would fail to suggest at first sight, are offered to us; but
would be an abuse of language to call them even hypotheses. I will ta

them in order: (i.) That the vagi are spent, or thrown out of gear. The suddenness of the attacks, both in onset and issue, seems against the opinion that these nerves are spent; thrown out of gear they may be. We know of many cases in which the vagi are thrown out of gear; as for example in bulbar disease, or under the pressure of growths or glands (cf. Pröbsting's well-known case), or in experiments upon animals; but in such cases the rapidity of the heart has not been by any means so great. It does not seem probable that abeyance of the vagi in man gives the heart play beyond 120 beats in the minute, or thereabouts. (ii.) That the vagi may be in abeyance, and at the same time the accelerators may be excited or vaso-motor centre affected. This suggestion sins against the economy of causes, and, as we must assume a close synchronism of disorder in each, we should be thrown back upon some cause behind them both. Some temporary change in the bulb might at once throw out of gear both vagi and vaso-motor governance; nevertheless such may be the case. (iii.) That the accelerators may be so stimulated as to overbear the normal vagi. It must be admitted that the onset and issue of the attacks seem in favour of some such supposition. In no experiment, however, has such a rate been obtained by accelerator stimulation; and reflex irritations of eccentric origin do not push the heart beyond 150 as an extreme limit. (iv.) That the cardiac ganglia are the seat of the disorder. But we find no changes in them which are inconsistent with a secondary origin; moreover, the latest researches on these ganglia by Dr. Gaskell and others seem to prove them to be remnants of the innervation of the arteries, and thus to have but a secondary importance in the cardiac functions. (v.) Some sudden change in arterial blood-pressures; this will not serve us, as the arterial blood-pressures are by no means constant, they are always rising and falling; any constant change of pressure would soon be compensated in the normal way: furthermore, while no ordinary tides of blood-pressure, as Mosso's experiments (Junot's boot, etc.) show, are competent to bring about so extreme a change of rate, there is no evidence of extensive areas of anæmia, as on dilatation of splanchnic veins, which might be adequate to call forth such phenomena. Were the heart abandoned to its own inherent rhythm its action would be irregular, which in tachycardia it is not, unless considerable dilatation has taken place. I confess that I leave my suggestion of extensive areas of anæmia somewhat wistfully, as a sudden expansion in the areas of the abdominal venous system would produce such results, as we see by the results of experiment and in exhausting diseases; still on this supposition the heart should not be distended, unless simultaneously the peripheral arteries are constringed. I think, however, the pulse is small in tachycardia, because by virtue of their tone the arteries fit themselves to diminished contents. We cannot, then, do more than guess whether the immediate causes of tachycardia lie in the cerebral cortex, in the mesocephalon, in the bulb, in the vagi, in the accelerators, in the cardiac ganglia or muscle, in anæmic areas, or in eccentric irritation, such as floating kidney (Balfour). Neuritis has been alleged as a cause; but there is no evidence of its presence, nor would it consist with the lo intervals of health. As the phenomena are remarkably uniform, t causation is probably not complex.

Of the immediate causes there is little to say. Over-exertion, dyspepsi mental shock or emotion, uterine disorders, auto-intoxication, loaded bowe any or all of these have been alleged with more or less hesitation. All th we can say, then, with any approach to certainty is that the victims of t disease are of the neurotic habit, and that in a few cases it has seemed be hereditary (Williams). It is alleged that Graves' disease, in which rapid heart is the chief feature, may be the instant result of an acu mental shock.

Sex.—The records of cases indicate that this factor has little or influence in the causation of tachycardia, the disease falls almost impartial on the two sexes.

Age.—In forty cases of Dr. Herringham's collection the age w recorded. In seven the malady dated from childhood; of these seven, fi were women. In twelve the first attack appeared beween the ages twenty and thirty; of these, six were men and six were women. thirteen cases the onset fell between the ages of forty and fifty; in thr the patients were over fifty when it occurred. Dr. Watson Willia reports a case in a man of eighty-one, in these attacks the pulse would le suddenly from 60 to 130. H. C. Wood reports a case as still recurrent in physician of eighty-seven years of age; the attacks began in his thirt seventh year; the onset is abrupt, and the pulse rises quickly to 200.

Diagnosis.—Tachycardia is an intermittent disease; if we rememb this we shall avoid confusion with other diseases in which a rapid acti of the heart is a more persistent feature. The larval form of Grav disease—a by no means uncommon form in which the thyroid is n enlarged nor the eyes prominent—may be distinguished from tachycardi in cases which have been watched for a sufficient time, by the long pe sistence of the rapidity. Moreover, in Graves' disease the action of t heart is more thumping. Fine tremor may be seen in many cardio-neurot cases, as may exalted reflexes also. Tachycardia is not a mere incide of neurasthenia. The pressure of a tumour on the vagi may be attend with a persistent rapidity of pulse. In cases of idiosyncrasy, cases which the pulse runs in the individual at accelerated rates, the persisten of the peculiarity will again decide the judgment against tachycardi and it may be added that in these cases, and in others of more less persistently quick pulse, the patient suffers less instant distre Cases are recorded on good authority in which the pulse of a pers presumably healthy habitually ran at 150 a minute. Binswanger h recorded such a case in a woman; in her the peculiarity had endured her life. I remember one day, when I was driving with a medical frien a man passed us on horseback—a fine-looking country squire in who there seemed no flaw; my friend told me to note him as he passe because his pulse ran habitually at 120. The owner of the pulse, patie I cannot call him, enjoyed fair health, but in the doctor's opinion woul be a "bad subject" for acute disease; this opinion he founded not only on a mistrust of the pulse, but also on a certain lack in him of resistance to fatigue and trivial ailments. Dr. G. Balfour, again (*Senile Heart*), refers to the case of a lady, then over seventy, who had had a large family and enjoyed good health, though of nervous temperament; her pulse had never been under 150. Of heart diseases the two to be excluded are dilatation and mitral stenosis. The tobacco pulse, if rapid (at first it is slow), is irregular. Alcohol, if it accelerate the heart's action, does so by inducing degeneration of the organ, and this lesion may be betrayed by its own phenomena. The accelerated pulse of cardiac dilatation is irregular. Old men who give themselves up to sexual indulgence have a pulse of increased rapidity, but tachycardia is not very likely to attack a man for the first time in old age. Fevers, diarrhœa, and other toxic or exhausting causes may be attended by a quick pulse, but such causes are not likely to be overlooked. In bulbar palsy the pulse is persistently changed; and if accelerated, is irregular and intermittent; in tachycardia the rhythm is even: moreover, bulbar disease has its own characters, such as faintness attended with a fear of death, a kind of "angina sine dolore." Finally, in none of these is the disease paroxysmal.

Prognosis.—Dr. Herringham thinks that after thirty years of age no patient of tachycardia is safe, and that few pass fifty. This, I think, is rather a darker forecast than I should be disposed to make. Much depends, as Herringham says, on the duration of the particular attacks and on the frequency of their return; if these last longer than five days the stress on the dilating heart leads to strain, especially in the elder patients. Two patients of mine are well past their climacteric, and to Dr. Watson Williams' patient of eighty-one I have already referred. In him, as in most patients as they advance in years, the return of the attacks is generally postponed; the intervals are longer, and there is more time for recovery.

Treatment.—Unfortunately this paragraph is but a short one—not because we have a prompt remedy, but because little or nothing seems to be of much service either in cutting short the attacks or in the prevention of them. As I have said, the attacks may get less both in number and severity with advancing years; and, perhaps, something can be done on general principles to make the system less susceptible to the causes of them, whatever these may be. That they lie in the nervous sphere the result of tonic treatment seems to indicate. During the attack tincture of digitalis in a little brandy is sometimes serviceable. The brandy I find is necessary, as in tachycardia the foxglove is especially apt to set up nausea. However, brandy or no brandy, it is often of little use, and patients soon give it up. If digitalis does not modify the rate of the heart it often causes diuresis; now in a heart quickened by the failure of intrinsic disease the drug often fails to produce diuresis, a result of bad prognostic meaning. One of my patients still clings with faith to a prescription of salicylate of soda and sodium bromide which I gave her ten years ago; she assures me that it is of much service to her in mitigating and shortening the seizures. I gave it on a strong hint goutiness in her family. This patient has had a fibroid tumour for ma years, but the attacks are certainly of still older date; there is no evide that the fibroid has affected her tachycardia in any way for good or e I recommend compression of the abdomen with a binder, but I th this method has not been well applied; a trained midwife should engaged to instruct the patient in the proper use of the bandage. Woo patient was relieved by drinking iced water and strong coffee, as if arouse reflex inhibition by the vagi. The application of electric curre of this kind or that, to the vagi in the neck, however promising at f sight, has disappointed those who have well tried it. Finally, it is s that a compression of the chest by the patient himself sometimes succe in stopping an attack. I have not had a good opportunity of putting t method to trial. It is to be essayed as follows:—The patient will thrust feet as hard as he can against the foot of the bed; then, pressing his ar closely into his sides, he will take a long inspiration; in the next pla closing the glottis, he will make a strong expiratory effort, thrusting h the while against the walls of the chest with the upper arms, and clasp them with the forearms. In this way it is said that the rate of the he may be directly controlled. After this fashion an old friend of mine u to cause his heart to intermit. During the intervals of quiescence p severing efforts must be made to nourish and invigorate the system. T digestion and the excretory organs are to be vigilantly watched a corrected, and all means are to be adopted to secure serenity of life an wholesome and regular occupation. One of my tachycardiacs began ride a bicycle two years ago, and with much advantage. Oertel's "he massage" seems to me to be no more than ordinary massage plus sugg tion; but massage is very useful in emaciated or podgy people, and, the more vigorous, Swedish gymnastics may be cautiously used w advantage. It will be remembered that any over-exertion or stress m bring on an attack; the treatment must therefore be trimmed betw the extremes of indolence and fatigue or sudden effort. A patient w rides the bicycle tells me that in this respect the bicycle is better than ho exercise; a horse may, and often does make a sudden demand on the rid nerve. The use of the graduated douche or of the wet sheet pro very useful in some cases; but for further particulars of this kind the rea is referred to other chapters.

BRADYCARDIA.—The reasons which justify us in retaining the na "tachycardia" make for the banishment of "bradycardia." Bradycar is a superfine name to denote slow pulse; it connotes nothing. literature a little pedantry may be harmless, nay, as a protest agai slovenliness may have occasionally its welcome side; in science it is pest. The name "bradycardia" is as pestilent as the rest because hoodwinks the student, who does not rid himself of the false preposs sion that in so large a word must lie a specific meaning; he does realise its emptiness. By neurasthenia we do not mean mere nervo debility, but a particular and definite group of symptoms of which nervous debility is but one feature. With the word "tachycardia" we introduce a new conception — that which I have endeavoured to set forth; with bradycardia we introduce nothing; the word is but wind. We know of no symptom group to be thus designated; bradycardia is slow pulse and nothing more.

Relatively to the heart slow pulse is a "functional" disorder when it is found independently of intrinsic and static lesion of the heart; thus slow heart in meningitis, terrible as is the disease itself, is yet a functional disease in respect of the heart. Slow pulse in fatty degeneration of the heart is not, in clinical language, a functional but an organic change. But, slow pulse connected with organic disease, whether in the heart itself or elsewhere, will not be discussed here.

The proposition that in all cases slowing of the pulse is due to the control of the vagus is not perhaps invariably true, though it is of very general application. I have said already that in a few cases slowing seems to be attributable, at any rate in part, to the muscle itself; but even then it is hard to say how far pulse retardation may be due to the vigilant nursing of the vagi. But in the case of certain poisons the muscular contractions seem to be slowed down directly, though even in them, as in fatty heart, it is difficult to share between the vagus and the muscle the function of each; whatever be the inherent failure of the muscle the vagus may and generally does intervene to spare it. In such states as senile broncho-pneumonia, where the tendency is to dilatation, the action of the vagus, whatever its immediate protection may be worth, turns to evil ere long even in the heart itself; as vagus action not only reduces the rate of the heart, which in itself might not lessen its work, but reduces the work also; and the organ cannot overtake its arrears. Therein lies dilatation, excessive internal stress and imminent strain. However, to leave these questions we have to turn rather to the slow pulse which depends not upon organic disease of the heart, nor indeed upon organic disease of eccentric position acting by reflection on the heart through the vagus, but to those functions, all perhaps following some reflex paths, which slow down a relatively healthy heart. Of these the following classes may be made :—(i.) Rise of blood-pressure, as seen, for instance, in its simplest form in the "expiratory diminution of rate"; or, conversely, in the temporary suspension of vagus action by continual sipping of a fluid: thus the heart's frequency may be raised twenty or thirty beats a minute (Waller). (ii.) Blood containing intrinsic poisons, such as carbonic acid or that of uræmia; or extrinsic poisons such as lead, tobacco, digitalis; or bacterial products, as in diphtheria, most of which act directly on the vagus or its centre, but some of which seem to affect the heart itself. (iii.) Reflexes from the irritation of eccentric derangements, such as those arising in the gastro-intestinal canal (dyspepsia, etc.), in the pelvic organs, in the throat or ear, and so forth. (iv.) The slow pulse of children. (v.) The slow pulse of hysteria, melancholia, and other psychical disorders. (vi.) The slow pulse of exhaustion, as after fevers or great fatigue (probably not reflex?). (vii.) The slow pulse of p The slow pulse of cerebral, bulbar, and cervico-spinal disease. (slow pulse of heart disease, disease rather of its walls than of valves, we have deliberately excluded from the section of functi disorders.) Epileptiform attacks (Stokes-Adams disease) seem, syncope, rather to be an occasional consequence of slow pulse tha cause of it; in uræmia the two events may spring from a common ca Vertigo and syncope are more frequent consequences of the kind; t are in my experience grave symptoms when associated with slow pu and suggestive if not conclusive indications of cardiac degeneration.

Again, in some persons an infrequent pulse may pertain to t normal state. I have never had my finger on the pulse of an epile at the earliest moment of an attack, but scores of times, as for insta in the wards of lunatic asylums, I have felt the pulse at the instan the seizure becoming manifest; I have never, however, found characteristic change in the rate. I find that Sir R. Gowers makes same remark. In the cases in which the association of slow pulse v epileptiform convulsion has been noticed it seems probable that the p retardation comes first, and that the intermediate factor is cere anæmia; that, indeed, the phenomena are those of convulsion on extr phlebotomy, the stage beyond deliquium. Of "normal slow pulse" we many examples; the most remarkable I have recently seen was in a vigor cheerful man who was in the Radcliffe Infirmary during the Michael examination for the M.B. degree in 1897. In this man a pulse of 28 c be raised on excitement to 32 or 33. Being a weather-beaten person over 60 years of age his arteries were not, of course, free from sign degeneration; but it was difficult to say that they were older than his ye Of the rate of his pulse in former years he knew nothing; he was unaw of it until we told him. I suspect that it had gradually come on a grew older. He felt quite well, and was vastly amused by our determ tion to find some grave mischief within him. He was admitted some trivial ailment, in order that he might be hunted well over by candidates, who, however, found nothing more to report; and Dr. S. W Dr. Mallam, and myself found him free from any other malady t that of eld. A pulse of 50 is no very uncommon rate in healthy pers rather in men, perhaps, than in women; in a friend of my own a puls 58, sometimes slowing down on fatigue to 54 or 55, has proved consis with great nervous and muscular activity up to years which are now m than mature. For him a pulse of 80 is fever; it never rises over 10 thereabouts, except of course under severe muscular exertion. Corvis record of Napoleon's pulse as habitually 40 is well known; Sir Will Broadbent has, I believe, recorded somewhere the case of an ath with a pulse of 36. Osler, who within the limits of his *Practic Medicine* rarely misses a point, tells us that physiological slow puls seen in parturition, whether premature or at term. The rate may dec from 60 to 44, and has sometimes fallen as low as 34. It is needles say that in all cases of alleged slow radial pulse the number of the car revolutions must be counted at the centre; as some of the waves may fail to reach the periphery. Some records of egregiously slow pulse can scarcely, one would think, have been of cardiac pulses. Roy used to say that a healthy heart might drop six beats and recover; but can a deteriorated organ cross such an abyss of time? We read of pulses of 20—nay, of 12 a minute; of stops of 15 seconds' duration—in one instance of an arrest of 30 seconds. An absolute stop of 15 or 20 seconds must surely mean fatal syncope, or epileptiform convulsion. Very feeble heart-beats may be inaudible even to the stethoscope. Fibrillary contraction is sometimes recovered from in animals, probably not in man.

All I know definitely about "hysterical slow pulse" I have found in von Noorden and Buchholz. If I have seen it I have made no note of it. For the variations of the pulse in mental diseases the reader is referred to the following chapters on these subjects. In respect of poisons we know that some of them, such as lead, may act indirectly by perverting the metabolism of the body, and thus generating intermediate poisons; uræmia and jaundice are often associated with a slow pulse. Most if not all these catabolic substances act, no doubt, directly on the vagi, centrally or peripherally. The poisons generated by bacteria—the infections—not infrequently begin by stimulating the vagi, so that the pulse is slowed; then the vagus is exhausted, the pulse quickens, and in the later stages is much accelerated—the mass of the blood being often much reduced in these diseases. In convalescence the cardiac centre seems unstable, and the pulse may be slowed or quickened by influences which in the normal state would prove indifferent. That muscarine slows the pulse is a familiar laboratory demonstration; and the accelerating effect of its antidote atropine is more familiar still. Tobacco, again, stimulates the vagi at first, and then paralyses them, or leaves them exhausted so that, in extreme cases, the heart is rapid and so irregular as to seem to be abandoned to its own rhythm. Rise of blood-pressure may retard the pulse remarkably; the fact is familiar to all clinical observers; but the rule that the rate of the pulse is inversely as the blood-pressure is open to many contingencies; it only holds when other things are equal: I think it better to put it that pressure is that part of the energy of the blood which is not turned into speed. The sum of the energy may be reduced. In the slow pulse of exhaustion the blood-pressure is often low; if vagus control be its cause the low pressure is due to the effect of this nerve in slackening as well as of slowing the heart; the residual blood in the left ventricle is more. I have seen this retardation fall to 45 in many cases of persons whose pulse in the normal state is of ordinary frequency. Some fifteen years ago, when very arduously engaged in practice, I was returning by night from a consultation in the west of England, when on leaning my head on my hand I felt my temporal arteries beating too slowly; the rate, then about 48, fell gradually to 44. I got a glass of hot brandy and water at Bristol Station soon after, thinking to mend my condition; but its immediate effect was to reduce the pulse, which had recovered to some 50 beats, again to 45. On the basis of this observation I have

supposed that the slowing of exhaustion is a protective effort of the v
which, in my case, were further stimulated in their gastric area by
brandy. After the brandy I fell asleep, and on awaking my malady
gone. It was attended with a sensation of sinking or depression ; and
times I have since recognised some abnormality of the kind by the sa
warning. During the last eight years, of a less harassing life,
derangement has altogether disappeared. Now here we had a b
breach of the rule that rate is inversely as pressure, for in my case
pressure was, as I have said, low ; and it rose as the normal rate
regained. The heart's output was probably increased.

Sexual exhaustion is efficient to reduce pulse-rate. But the ot
day a patient was sent to me by a distant medical friend who h
found in him a slow pulse, about 40, attended with a sense of dep
sion, almost melancholic, especially of a morning. It was a great eff
for him to get up to breakfast ; although after he had got to work
play the sensation wore off. At the times of slow pulse the temperat
also would fall to 95°. He was in business, but in an easy one ; he had
cares, his habits appeared to be correct, and he had had no troubles.
was fond of physical exertion, and could and did ride, shoot, and
forth even to the full, without being the worse. His age was forty.
examination of his heart nothing abnormal was to be found. His o
medical man had cut down his tobacco (usually $2\frac{3}{4}$ ounces a week) w
advantage, but without much relief. I ascertained that he gave him
up to excessive marital intercourse, even to daily indulgence. My p
scription was a separate bedroom, which will probably work a cure.

In some cases of temporary slow pulse with "nervous exhaustio
the voice becomes hollow or even feeble. In one case I remember
patient, partly in timidity perhaps, intimated that he was too mu
exhausted to do more than whisper a brief reply. It is possible t
some of the cases of slow pulse in children are due to self-abuse ; but
no means all. To find a pulse of 50 or 45 in a little boy or girl used
frighten me no little ; I regarded them as the barbarians regarded
Paul. But as, often enough, nothing happened I gained heart ; and
now, if still on my guard, not prophetic of evil. In some cases wor
may be the cause of the retardation ; but antidotes for worms do
always prove the connection. Nevertheless, as some arrhythmia may
present, and perhaps some heaviness or drooping of manner may
exaggerated by anxious parents, these cases are not a little embarrass
for a few days. Gastric catarrh, again, is among the causes ; and pr
ably in the child the heart centre, like the temperature centre, is m
susceptible than in later years. The ages of such patients run fr
four or five to fourteen or fifteen. The child may be languid and out
spirits, or dyspeptic, when the state of the pulse is found out, as it we
accidentally. Irritation of the vagi is again the probable explanatio
indeed, this seems to be the first factor to be thought of in all cases
slow or intermittent pulse, yet it may not be the invariable cause. Sl
pulse children are usually of neurotic constitution.

The slow pulse of convalescents from fevers and other exhausting diseases, is a common event, and is sometimes suggestive of cerebral complications, especially in children; it is probably due to vagus irritation, set up, it may be, by carbonic acid or by some toxin. Or the cardiac muscle may be poisoned. Thus I have seen it in severe bronchitis with distended right ventricle, much residual blood, and greatly overcharged veins. Intermittence is seen in these cases also, which may point to vagus protection.

The slow pulse of pain is a phenomenon full of interest: it must be due to reflex stimulation of the vagus; thus it can readily be produced by experiment; and it is not unfamiliar, under the like conditions, to the practising physician. Sir Richard Powell mentioned an interesting case of this kind at the meeting of the British Medical Association in 1894. The patient was subject to neuralgia and to palpitation, but not together. An attack of pain would stop the cardiac disturbances. Sciatica is perhaps the pain most efficient in producing this result; but almost any sudden paroxysm of pain of sufficient severity may be reflected in the pulse. Its chief interest lies in its bearing on the causation of angina pectoris, whether of the graver or of the "functional" kind. Whether slow pulse may ever be due to a failure of the accelerants we cannot tell; in the cases of "exhaustion" above described such may be the case entirely or in part. Of the intimate relations of the intra-cardiac ganglia to the functions of the heart we know little, or indeed nothing; Dr. Gaskell regards them rather as survivals of the nervi vasorum than as dominant factors in mammalian cardiac evolution.

As bradycardia denotes a symptom and not a disease, or as, in other words, it signifies no more than a phenomenon common to many definite groups of symptoms, and as there is not, as with tachycardia, any peculiar group of which it is itself the main or central feature, it can have no diagnosis or prognosis. All that can be said is that it may depend upon irritation of the vagus only, the heart being sound. In such cases it will often, of course, be associated with arrhythmia and intermittence. Such conditions are usually curable by removal of the causes, and especially by careful mastication of the food. Momentary efforts often aggravate the condition, but in a sound heart persistent exercise removes it for the time. It is usually worse after meals, and is attended with flatulence. The urine must, of course, be minutely and repeatedly examined in all its qualities, and signs of cardio-arterial degeneration duly appraised; remembering however that, if due to degeneration of the coronary arteries, the most usual organic substratum of slow pulse, signs of disease may be absent or very indefinite. But we cannot pursue these parts of the subject; from what has been incidentally said the reader will know where to turn for descriptions of the symptom groups to which slow pulse is subordinate. Under these several heads will also fall the means of treatment, if slow pulse can be said, any more than cough or dyspnœa, to have any treatment of its own. Static disease of the heart apart, slow pulse needs not even palliative treatment; it no dangers of its own.

It is impossible to give any list of references in respect of a m symptom such as slowness of the pulse. The reader will find two rec articles on the subject in the *Lancet* of 30th January 1897 by Dr. J Ogle, and one by Professor Osler in that of 27th February 1897. these articles, however, the symptom is chiefly regarded as significan intrinsic cardiac degeneration.

SYNCOPE.—Whether the heart stops altogether in syncope is unknown; it probably beats with a beat so feeble as to escape our sen It may be arrested, but it seems impossible that the heart should arrested during all the span of a long faint; I have said elsewh that Roy, on the basis of large experimental observation, thought t the heart certainly may drop six beats, possibly more; but that bey some such number as this there is great danger of death. Yet when are discussing the ordinary fainting fit these calculations of more or l around the margin of the grave seems fanciful: "No one dies of a fai one may say; or another may say with equal truth that sooner or la almost every one does. Yet the syncope which cuts the vital thr at the end of most fatal illnesses is evidently something so different degree and contingency from the ordinary faint of the ladies who carried out into the vestry, that here we must fix our attention clusively upon the functional disorder. The church faint is not prima a cardiac failure, but an expansion of cutaneous and splanchnic ves with fall of arterial pressure.

Yet of this curious disorder no full explanation is forthcoming, surm as we may. It is a very common malady; perhaps no woman pas through life without experience at least of its premonitory symptoms. faint is not the exclusive privilege of woman; every physician has s men fall like oxen—for instance, in the gallery of an operating theat A very sturdy and stout-hearted man once fell suddenly to the floor my consulting-room, where a moment before he was complaining to me some temporary disorder; partly dyspepsia, partly fag. I have kno him for some quarter of a century since that day, and, so far as I aware, he has never fainted since. Again, an old friend of mine, the young man of some five-and-thirty years, then and since hardy a sound, on rising suddenly from bed in the middle of the night to em his bladder, fell backwards, drenching himself with the contents of t chamber-pot. His wife told me that he lay unconscious for a "minute two." The anxiety in such a case is whether the attack were a faint an epilepsy: the circumstances of this attack, chiefly the person's sudd uprising, pointed rather to syncope, and time seems to have ratified t opinion, for no such attack has reappeared. On the other hand, syncope not usually an isolated event in the life of the patient. People who fai are, as a rule, "given to fainting"; such persons dread hot rooms a congregations where the distribution of the arterial blood may wide oscillate. Or, again, they dread certain strong sense impressions—such as the sight of blood or strong odours, which may inhibit the heart; Italian women are said to be peculiarly liable to faint on the smell of flowers. On one occasion I was dining with a charming hostess who had decked her table with charms like her own: as we sat down, one of her guests, apologising for his weakness, said that he should faint if he sat with his back to the fire, and at some sacrifice of harmony he was conveyed to another seat; no sooner had he been dealt with than another guest thought he had better add that he himself was subject to faint in the midst of a strong scent of flowers, and that he also had better mention his unhappy susceptibility in time. There was nothing for it but to clear the table of the spoils of the Riviera; after which twofold commotion things fell a little flat. Both these men were literary men of more sensitiveness, perhaps, than virility, and had better have stayed at home. In such persons, of either sex, the pulse varies too widely on quickly rising, sitting, or lying down. The limits of such variations should be within five beats; if they are wider, and they are often as wide as twenty beats or more, the compensatory mechanism is defective.

Syncope without any organic disease may be fatal; such cases are not extremely rare; they are common enough to give a colour of caution to prognosis, and of care to the treatment. In my experience of such sad events I am disposed to think that the faints due to agonising pain are more likely to be fatal than those arising from sudden displacements of blood-pressure. The inhibitory effect of intense pain may, it would seem, arrest the heart through the vagus to a degree incompatible with life. Death in angina pectoris is due to this reflex effect of pain; the pain, in my opinion, having its seat in the aorta.

The premonitory symptoms of fainting are known to every one. He is a fortunate man who, in the weakness of some acute malady, influenza or the like, has not been conscious of the swimmings and exhaustions which may usher in a full attack. If some of us have never fainted, we have all of us felt faint. When the attack is fully established unconsciousness is complete, the respiration is only to be detected by the use of a feather or a mirror, or not even thus; and the pulse, cardiac and arterial, is likewise imperceptible. If the urine or fæces are voided, it may be said with some certainty that the attack was worse than a faint.

Whatever the remoter causes, such as general anæmia and debility and the rest, the immediate cause of fainting is encephalic anæmia. The same is true, of course, in organic diseases, such as those of the heart. It is the first duty of the physician, as it is the care of nature herself, to place the patient in a position to favour the return of blood to the brain; the head must be dropped even lower than the trunk of the body. As on the one hand Junot's boot will produce syncope, so on the other to elevate the legs will aid in its dissipation. The blood-pressure must also be raised by causing contraction of the superficial blood-vessels; cool air, and the admission of it to the skin by unfastening the bodice, is one means of attaining this end; and it is, no doubt, of some use thus

to loosen any bands which may be hampering the respiration ; a d
gasp, if it can be obtained, stimulates the heart to contract by unload
the right ventricle. The respiration is called upon by reflex stimula
also, such as smelling-salts, dashes of cold water, and so forth. In ca
of anæmia compression of the abdominal veins may be useful, or
application of an Esmarch's bandage to one leg or both legs, and
extreme cases artificial respiration, or even transfusion of blood, mi
be needed ; but such difficult means are fortunately rarely if e
necessary in the functional cases which alone are under our discuss
in this place. It is desirable, perhaps, to add that after the restorat
of consciousness the physician should not leave the patient without
strict caution against the resumption of the vertical position until
tendency to a return of the attack is averted. For a fuller discussion
the physiology of events of this kind the reader is referred to an arti
on the circulation of the brain, which will appear hereafter.

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to Dr. Herringham's article.

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T. C. A.

MECHANICAL STRAIN OF THE HEART

STRAIN of the heart, it need scarcely be said, is not a malady, but the cause of maladies, both of this organ and of the aorta; possibly, also, of arterial disease beyond the aorta (Traube); in respect of this last suggestion, however, the evidence of an affirmative kind is as yet scanty and uncertain. We shall see presently that to measure stress as a factor of heart and aortic disease, with any approximate accuracy, is beset with no small difficulty. That stress—mechanical stress—is an important factor in disease of the heart no experienced observer can doubt; moreover, as we shall find, in certain acute cases of strain this factor can be indicated with some precision: in chronic cases, however, stress is so intimately confused with other factors—such as the abuse of alcohol, the infections of rheumatism or syphilis and the like—that it is often exceedingly difficult to distribute its due weight to each one of such factors. For example, many most useful observations concerning strain of the heart have been made upon soldiers; yet there is perhaps no class of persons in whom the various factors of cardio-arterial disease, including improper dress, are more difficult to estimate severally. On the other hand, however, the part of stress in the causation of heart diseases comes out plainly when we consider such cases in numbers large enough to eliminate, or rather to reduce, the risk of error;—when, for instance, we contrast large numbers of persons engaged in laborious callings with large numbers of those whose pursuits are mechanically less urgent,—when we compare forgemen, h men, navvies, wharfingers, Cornish miners or Tubingen wood-cutters, v have no monopoly of vice, with clerks, professional men, or even w persons whose callings are in the open air, but not to heavy musc exertion. The part of stress, mixed as it still is with other factors made evident, again, in the comparison of the cardiac affections of m with those of women and children. In any case, while we remem that, relatively speaking, the function of every heart, healthy or diseas is concerned in muscular exertion, yet when we enter upon a discuss of strain of the heart we are understood to refer to the effects of muscu exertion upon hearts which before the strain were either strictly virtually sound. At the same time, we shall not forget that unus exertion too often brings out a latent defect in a heart which un ordinary circumstances, and for some time at any rate, would have pass as sound. In men beyond middle life a breakdown of the heart is oft thus acutely determined. In most cases of angina pectoris, sudden appearing in persons previously regarded as healthy, some unus bodily effort determines the first attack.

For clinical purposes strain in its effects upon the heart may co veniently be divided into functional disorders, injuries to the cardi muscle, and injuries to the orifices and valves of the organ,—chiefly to t aortic. It is apparent at once that this distinction is a superficial on mechanical disorders tend to become permanent, and aortic disease especially in the long run, are apt to be associated with muscular faul Still, the distinction may be admitted for clinical purposes; and d orders of the first class have been considered in the chapter on "Fun tional Disorders of the Heart," p. 821; those of the myocardium will in the chapter devoted to this subject in the next volume; and those the third class among the valvular defects. Without some such divisio the subject of heart diseases would be almost unmanageable.

When Harvey announced that the circulation of the blood belongs the sphere of mechanics he wrought a revolution in physiology. Und his teaching vague and fanciful apprehensions gave place to more positi conceptions. From the time of Harvey, although physiologists have n asserted that mechanical conceptions can cover the whole phenomena the circulation, they have learned to see, nevertheless, that these conce tions cover so much of the ground that in mastering them they and the children may find reward enough. If this lesson be not thorough learned it has gained a good hold, and is proving its fruitfulness; y it is not till the days of Marey, Ludwig, Roy, and Gaskell that we fi a serious endeavour to ascertain the order of the phenomena of t cardio-vascular apparatus as a machine, and to indicate the limits of i physics in the direction of those nervous agencies which can only called mechanical in a forced acceptation of the word. For a full d cussion of cardiac physics, however, I have the advantage of referri the reader to the chapter on this subject from the hand of Profess Sherrington (p. 464).

Cardiomotive force is equal to the output of the heart plus the resistance to the travel of the blood in the vascular system; a resistance chiefly due to friction, or, in other words, to the viscosity of the blood and the diameter of the channels through which it runs. The elasticity of the arteries adds nothing to the cardiomotive force; by it some considerable part of this energy is stored up in a potential form during certain moments of the revolution, to be given out at other moments. The elasticity of the arterial tree diminishes from youth to age, and as it is lost the work of the heart is increased; the work of the heart is thus increased at a time when the powers of the body are on the wane: but it is so difficult in later life to distinguish between lesions due to variations of stress and those due to intrinsic degeneration in the texture of the viscus, that when we speak clinically of strain of the heart,—that is, of a permanent "after-strain" or "set" towards other than the normal lines of its action, or of a permanent loss of capacity within these lines,—we are understood to contemplate young or comparatively young subjects, to contemplate premature tensile or shearing strains in the causation of which degeneration has had but a small initial share or none.

In what way or ways may stress in the heart produce strain? What are the conditions of abnormal pressure within or about the organ? How does it adapt itself to unusual stress? In case of failure where do the effects make themselves felt?

In the first place, we must realise that while, on the one hand, the arterial blood-pressure is incessantly oscillating, yet ordinary changes of stress do not raise blood-pressure permanently. If I lift a weight, say of ten kilos, my blood-pressure will rise promptly, even by some 20 per cent. After a few seconds or minutes, however (the interval depending on incidental circumstances), the blood-pressure will have returned to the initial level. Again, I may constrict a large artery, even the aorta, or by injection I may increase the whole mass of the blood in the body by as much as 20 per cent, yet in neither case will the blood-pressure (by "blood-pressure" the mean arterial pressure is usually signified) present more than a temporary rise. This is not the place to enter into the wonderful mechanism—none the less mechanical that the nervous system is largely concerned in the balance—by which these adaptations are made, the heart and aorta saved from strain, and the various areas of the body protected from irregular afflux of blood: suffice it to say that the readjustment is largely determined by reductions of resistance. But there is another factor, the factor of output; if the output of the left ventricle be increased, and this increase be not compensated, as is usual, by a fall of peripheral resistance, the chamber will be under increased stress, and may suffer strain.

Seeing then that, however transiently, blood-pressure is raised by muscular effort, and that output likewise may undergo considerable and even extreme variations, are the compensatory mechanisms always adequate to readjustments so rapid and so complete as to make the notion

of injury to the heart under ordinary circumstances improbable? If
decide that the evidence points in this direction we may infer, never
less, that an unhealthy heart, or one subject to other adversity, will su
under great exertion if these compensatory mechanisms fail, or are
sufficient.

A series of experiments upon the blood-pressure of persons enga
in muscular work was projected for the years 1895-96 by the late Profe
Roy and myself, but my colleague's unhappy illness prevented this
other investigations. One rather curious fact, however, seems to c
out in the course of the more or less desultory observations which I
others have made upon athletic men in Cambridge and elsewhere, nam
that in them, as a rule, the habitual blood-pressure ranges low. A
observations were made with Roy's sphygmometer upon men given
arduous muscular work. These observations, taken at times of comp
or comparative rest, seemed to indicate that in them the arterial press
ranged habitually under the average. In my own person Alpine cli
ing and, in later life, cycling have always been followed by a fall
blood-pressure. It is hard to say what happens during spurts or at
outset of an excursion, but very soon afterwards the pulse not o
quickens but softens; for the rest of the day and night, at any rate,
pulse is soft and dicrotic. If on account of bad weather I cannot t
sufficient exercise, my pulse gives me the sense of higher pressure, an
am conscious of a falling off in vigour and temper. I am well aware
the difficulty of measuring the blood-pressure in man, and for the m
part we have as yet to be content with the impressions of experien
clinical observers whose impressions must be taken for no more than th
are worth: still such impressions are not without value. If a num
of observers skilled in the pulse agree that the radial pressures of a
of men seem to them to be low, this agreement is worth consideratio
at any rate nothing better is to be had except a few records with Ro
sphygmometer, which corroborated those of the finger. An interest
passage in Dr. George Oliver's treatise on Pulse-Gauging came under
notice as I was correcting these pages for the press. He says (p. 12
"Observations with the pulse pressure gauge have shown that, wh
other indications are favourable, the lower ranges of pressure are
only more salutary, but are very often compatible with the highest healt
Since these words were written, Dr. Tunnicliffe, in conjunction with
Brunton, from Mosso's laboratory has published like conclusions;
so likewise have Tangl and Zuntz.

The converse of this proposition is seen in the rise in blood-press
in advancing years observed not by myself only (3), but by such exp
enced physicians as Dr. George Balfour.

Habitual muscular exercise, then, tends in the main not to raise, but
reduce mean arterial blood-pressure; or persons with relatively
pressure may be well adapted to such exertions and naturally take
them. During the first stages of muscular exertion, no doubt, the ra
of blood-pressure is high; perhaps throughout severe exercise the m

may be above normal. But during steady work it probably falls at least to the normal mean, and during rest and on quiet days may range below the average standard in sedentary men. If this be so, the hearts of athletes and of ordinary labourers should be, not at a disadvantage, but positively at an advantage.

How are we to reconcile these apparent contradictions? In one breath we say that excessive muscular exertion may damage the heart; and in the next, that on the whole the stress on the heart in muscular men is not more but perhaps less than in men who lead more sedentary lives. For while on the one hand I note that the blood-pressure of athletes runs a little lower than the average, on the other I note that the blood-pressure of men who lead sedentary lives, without denying themselves a like abundance of food, often runs high. I venture to think, from some little experience, that in members of a university or of the learned professions the blood-pressure tends to rise as athletic habits are laid aside. Perhaps by abstinence this disposition may be prevented; but I am always assured by brain-workers, and I share the prejudice, that for them also a somewhat liberal diet is required. For my own part I have found that I crave for food more when using my brains from day to day in my study than when taking vigorous exercise in the open air.

We have arrived, then, at the paradoxical result that muscular exertion tends on the whole to lower blood-pressure, and a sedentary life to raise it; yet that certain diseases of the heart are to be attributed to the mechanical effects of muscular labour. To reconcile these opposite positions we may make a twofold reply: although the mean result of muscular exertion may be to reduce arterial pressure, yet the initial effect of such exertions is to raise it, often enormously. If we may make the assumption of a man steadily working with his muscles at a uniform rate without rest the pressure in his arteries would probably be slightly under the mean of ordinary citizens; and although the rate of the heart would be increased, the total daily output might not be increased: if, on the other hand, we assume that the same man carries eight bushels of wheat up a flight of steps every ten minutes, although the mean of his blood-pressures for twenty-four hours may not be very excessive, the maximum pressures, that is, the initial rise at the outset of each effort, may be very high. Again, let us suppose that this man does not carry sacks hour by hour and day by day, but that he is engaged as a checkweighman and takes a sack up occasionally; it is likely in this case that his maximal arterial pressure, as he shoulders the sack, will be higher than under the same stress more regularly undertaken by a porter whose respiration, blood volume, and vascular distributions are better adapted to the recurrent stresses. Yet the weighman may be more or less accustomed to labour, and, if not used to such efforts as the porters are, he is nevertheless in something like training; if, however, a clerk from the office were fired, by emulation of the porters, to carry sacks, the absence of habitual adaptation to such exercises might cause so sudden and relatively so great an increase of arterial pressure as to rupture a limb of the aortic valve.

In what, then, does such adaptation consist? Partly in the behavi of the skeletal muscles; partly in the function of the respiration. take the muscular system first: we readily and rightly understand t the first effect of a general contraction of the muscular system must to compress the vessels embedded therein, and thus at first to raise blood-pressure to a degree answering to a partial closure of this v area of the circulation. Marey demonstrated with the sphygmogr that even to throw the muscles of the legs into spasm (while breath freely) raised the arterial pressure considerably. But in the next pl such is the exquisite provision of nature, the blood-vessels, under reflex influence of the afferent nerves of the muscles, or, it may be, un the influence of an increasing acidity of their lymph when in acti dilate, and, reopening the vascular area which was momentarily c stricted, they flood the muscles anew with arterial blood; thus at o the muscles are fed for the work and the peripheral resistance is lower This afflux is independent of the general arterial blood-pressure. Ev under passive exercise also (massage), as Mitchell, Brunton, Tunnicli and others have shown (vol. i. p. 378), the flow of blood through volunta muscles becomes more abundant; and thus blood-pressure is reduc if the kneading be unattended with irritation of the skin, whi raises blood-pressure. To this compensatory mechanism we must add, most cases, an increased circulation in the cutaneous area and sweatin as we see in the major epilepsy. If the blood-vessels, by the deteriorati of advancing years, or of poisons such as lead, alcohol or syphilis, be l lively; if the blood be more viscous, or deficient in oxygen; or if aft some disease the nervous machinery be less sensitive, less effective, suffer any other disadvantageous change, the muscular reservoirs may op more slowly or less completely, and the arterial pressure will not fall readily to the normal mean; the heart may not get the relief which its due, and this organ and the larger arteries may suffer strain: again, the output of the left ventricle, increased probably in any case the first acceleration of the rate subsides, may continue in a greater ra than the fall of peripheral resistance, and the mean blood-pressure may continuously higher than during rest. Dr. Weber, during an ascent the beginning of his holiday, stated the initial rise of his pulse-rate be from 74 to 122; but after a week's active walking the rise was fr 74 to 105. He also noted that at first the systolic sound was shorten and less distinct (the systoles probably being "fractional," and the residu blood on each contraction large); but, as training advanced, the sy tole betrayed less interference, or even improved upon the quality of i tone before training began. Some cardiographic tracings taken on man in severe exercise indicate at first great rise of blood-pressure; t upstroke is much higher, and systole encroaches more on diastole. the impulse quickens, although the percussion is still powerful, t summit of the curve becomes sharper, the ascent more upright, and t duration of systole less extended.

Once more; an abundant supply of blood to the muscles, whatev the remittent checks of the actual muscular contractions, brings about, after the initial moment, a large increase in the mean volume of the flow. What becomes of this abundance? Will it not try the heart at first in another way by flooding the right side of it, and thus throwing stress upon another part of the organ. This danger is, I think, as great as the rise of pressure on the arterial side; when the functions are duly adjusted it is counteracted by the capacity of the muscular and pulmonary systems, which may not only hold nearly all the blood of the body, but are less liable to be embarrassed by incidental adversity: moreover, Dr. Oliver has shown that as muscular exertion goes forward a considerable transference of juices from the blood-vessels takes place into the lymphatic areas. Nevertheless, engorgement of the right side of the heart is an evil to be counted with, and one which happens under exertion more often than we are disposed to think; and thus, unless the output be enlarged, the residual blood is excessive in one or both ventricles. As arterial blood-pressure falls venous pressure may rise, and the pulmonary artery and right heart may be fatally distended. In elderly people, whose lungs may be emphysematous, grave heart disorder may come about in this way; but in the young and vigorous the heart, though, as I have said, it often dilates until it beats in the epigastrium, soon recovers itself as the lungs expand and the blood is redistributed. But if the exertion be both hard and long continued, harm may be done even in the young, especially in boys; the more so as fatigue products are passed into the blood. Hence it is that prolonged efforts, such as paper chases and the like, are bad for boys, and murderous to the middle-aged. Such a case I have seen recorded in a man who, at the age of 46, took to a tricycle, and after a brief apprenticeship rode from Brighton to London (53 miles). The physician who was hurriedly summoned on his arrival in town, found him faint, with a pulse of 141, and cyanosed; the cardiac dulness was extended a quarter of an inch to the right of the sternum, the apex beat being in the 6th interspace in the mammary line.

That the respiration is an important factor in the blood-pressure, and in the run of the circulation, is apparent to every one who has watched the traces of the kymograph. Dr. Waller, Professor Tigerstedt, and others have carefully discussed the effects of the respiration on the functions of the heart; and Dr. Morison has recently drawn the attention of physicians to them again in the pages of a medical journal (58, p. 966).

That the respiration is quickened in exercise is a matter of constant experience. Stimulation of the peripheral end of a muscle-nerve produces considerable increase of respiratory movement, even when the muscles affected have been removed from the sensorium by cutting the sensory paths. The muscles may manufacture something which, reaching by way of the circulation some nervous element in the respiratory mechanism, stimulates it; the respiratory centre in the bulb may be thus stimulated. Professor Sherrington tells me that Zuntz and Goppert have proved that this something is not CO_2; nor again a deficiency of O.

Thus some waste product of the muscles seems to excite the respirati to greater activity, while at the same time larger quantities of blood a being injected into the venæ cavæ; how large this quantity is may guess when we remember that on the contraction of a muscle blood-vessels open out so widely that it can contain at least one-thi more blood than when at rest; even when at rest, the skelet muscles hold something like a quarter of the blood in the body. To th forcing of successive charges of blood by the muscles beyond the valv of the veins we have to add the suction of the respiratory movements.

The most important condition in the filling of the heart durin diastole is of course its own previous contraction; but how far the hea itself exercises suction upon the blood as it enters is a problem which, yet, is far from being solved. In the well-known experiments of Gol and Gaule, negative pressures in both right and left ventricles we recorded in dogs, ranging from 100 mm. of water in the left, and fro 10 mm. in the right, to numbers between 300 and 400 mm. It is not y known, however, what the negative pressures in a strongly acting huma heart may be. Prof. Tigerstedt, in his new work on physiology, says th the conditions of an effective suction are so many and complicated that present no accurate opinion can be given on the matter.

As regards respiratory pressures, it is obvious that the pressure the atmosphere on the extra-thoracic veins must be greater than tha which, through the lungs, can be exercised on the veins within the chest thus these veins and the heart must be distended in proportion the difference. In inspiration this difference must be increased: th negative pressure within the chest must be increased, and in some pr portion to the depth of the inspiration. Hence the aid of orthopnœa venous retardation. The intra-thoracic veins, the auricles, and the pu monary artery must be distended, and the circulation would cease if sufficiently deep inspiration were held on. In expiration, on the othe hand, the negative pressure in the chest falls, and the access of the bloo to the thoracic veins is slackened. Even the systole of the heart itself, b which movement much of the blood is driven out of the chest, must exerci some influence in the direction of suction towards itself, or, more accuratel towards the great venous reservoirs. Now, whatever values we put on the several factors, we perceive that violent exertion must be attended by considerable oscillation of pressures in the thoracic veins and right hear oscillations due in part to the temporary rise in arterial pressure on th initial compression of the intra-muscular vessels, in part to the subsequen afflux of blood from the expanding muscular areas, and in part to the var ing negative pressures of the respiration. The thick-walled ventricles an the aorta in which blood-pressure is high "will be least influenced, an the right auricle and the vena cava, which are thin-walled and almost zero pressure, will be sensibly affected, the amount of blood-flow to th right side of the heart will be practically determined by it, and the le side will rapidly be affected in its turn. The left side will then drive large quantity of blood forward soon after inspiration has begun, a smalle quantity soon after expiration has begun. A violent and prolonged expiratory effort with closed mouth and nose may even cause a temporary arrest of the circulation, the intra-thoracic vessels being distended and the auricles unable to contract. Conversely, an expiratory effort made in the same way may arrest the circulation, as the venous blood cannot enter the compressed right auricle" (Waller). Now we know that on sudden and violent effort the chest is often fixed and the glottis closed. Again, in prolonged exertion, such as hard and long running, the advent of products of waste into the blood—sarcolactic acid, it may be, and others—must not be forgotten; for if they do not tend directly to increase the stress on the heart, they may do so secondarily and relatively in so far as they weaken the muscle of the organ. The rôle of each of these factors in the play of muscular exercise upon the heart cannot as yet be distinguished, still less calculated.

The clinical features of heart-strain, broadly speaking, are not so obscure as their causation. In my first paper on heart strain (1), I stated my opinion that dilatation of the right side of the heart is an early effect of prolonged exertion. The effect of sudden stress tells rather on the aortic area; that of more prolonged exertion, such as running, hill-climbing, or steady rowing, rather on the right heart. Time has strengthened me in this opinion; and Roy and Adami, Oertel, Dr. James Barr, and other authors have given their support to it. Not only are we strengthened in the opinion that dilatation of the right side of the heart is an occasional consequence of these prolonged exertions, but I now believe that dilatation of the right chambers is a frequent, I had almost said a normal incident of such exertion. I shall not be surprised to learn, from Röntgen rays or otherwise, that dilatation of the right heart and pulmonary artery is a common and transient feature in the adaptation of the heart to the variations of its work, especially in youths. The development of the muscles of respiration and of the lungs to capacities far beyond those of ordinary life, is a condition of training far too little understood or sought after, and takes a considerable place in the amendment of those selected cases which benefit under the Stokes, Oertel, or Nauheim methods. The safety-valve action of the tricuspid valve (Wilkinson King), and the apparent provision against this distension in ungulates by the moderator band, demonstrated by the late Professor Rolleston, are not to be forgotten in this connection. However this may be,[1] that dilatation of these chambers and secondarily of the left side also is a common result of prolonged exertion, and that it is often aggravated by the disabling effects of the circulation of waste products of a "curarising" kind, or by the nervous exhaustion of great fatigue, is tolerably well ascertained in a broad if not very accurate sense. The dilatation is, I think, concerned in "second wind"; the healthy heart increases its output, the lungs expand, resistance falls, the

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[1] The dilatation of the left ventricle, although it certainly occurs, cannot, I think, be satisfactorily explained with our present knowledge. Probably it is due to loss of tone and large residual blood, or may be due in part to some nervous sympathy.

right ventricle pulls itself together, and second wind is established. T
process, trying enough to an unsound or defective heart, to young bo
and to elderly men, is to the healthy heart of comparatively you
adults perhaps never injurious; I have many times seen undergradua
and others look ghastly at the end of a long spurt of hard exercise,
I never saw a sound young man the worse for a temporary stress of t
kind: if, as in a few cases which I have seen again and again in grow
youths, dilatation of the right heart occurs, leading to cyanosis, panti
and confusion or vertigo, this oppression is generally sufficient of itsel
stop the exercise in time. Even in children, whose frames are immatu
and who are apt to be overdone by prolonged stress, how rarely is the b
strife of hooping-cough attended with any ill consequences to the he
In a few cases, however,—in untrained men hard driven by haste
peril,—prolonged effort, exhaustion, heart stress, and fatigue produ
come in to complicate the reckoning, and persistent harm may be done.
have already published one carefully observed instance of cardiac dilatat
in my own person (1), I will now describe another. Some fifteen ye
ago I was called in the middle of the night, when no doubt more or l
fatigued already, to take a mail train at a station about four and a h
miles distant; when I had hastily dressed, I discovered that the fool
cabman who had brought the message had driven back to town. In fo
minutes I had to catch that train; and, running all the way on
hilly road, I did catch it. Profusely perspiring, I stripped the instant
sprang into the carriage, and found the transverse dull area of the he
considerably extended, as it was on the Dom in 1869. The radial pu
was rapid, of small volume and low pressure; I felt a little sick, and
face was cold. After a good rub down and an hour's rest in the trai
was quite restored, the borders of the cardiac area had receded, a
I felt no more of the stress. But it might well have been otherwise;
would have been otherwise if at that time I had been in bad conditi
It is thus in persons at and after middle life that the physician has
patch up the heart thus strained; some of these patients recover af
months of disability, others never recover, though life may continue
some years. An old friend of mine, when about fifty years of age, th
strained his heart by hard walking in hot weather on the Italian side
the Alps. He broke down and came home, when we found the dulness
the heart much extended transversely, and other signs of dilatati
The pulse was extremely irregular and intermittent, and these charact
it never lost, though some fifteen years of a valetudinarian life remained
him before œdema and albuminuria ushered in the closing scenes
his life. I have notes of many such cases of strained heart, especia
in men who by years or by frailty were passing or past their prin
Pain and constriction are felt in the acuter cases, but rarely (or nev
shooting into the arms; though it is sometimes felt as far as the second
intercostal space. There may be a panting dyspnœa, a cold dew on the fo
head, yawning, and exhaustion. The pulse is proportionately irregular
force and rate, and intermittent (vagus protection). A man of lett

whose constitution had been shaken by profuse hæmorrhages in early life, took to the bicycle in middle age, and often rode hard and far. He complained to me that at times he felt some discomfort from it. On careful examination I found no sign of disorder; but I begged him to end his next hard ride at my house. I then found his heart irregular and intermittent, the arterial pressure low, and the right ventricle dilated. Fortunately on his next visit he was well again, but repentant. In the worst cases œdema of the bases of the lungs is found on the following day. Such attacks pass off hardly and slowly; the pulse long remains irregular and feeble, and the breathing embarrassed by the least effort. There is probably a large quantity of residual blood in both ventricles for a longer or shorter time, the signs of dilatation appear on the left side also, arterial pressure falls, and the mitral orifice may yield. Such a patient may, indeed, fulfil the duties of a tranquil existence for some years; but he may remain languid and pallid, unfit for much physical exercise, and in all the work of life soon wearied into fretfulness and depression of spirits. In the next stage of the disease albumin appears in the urine, and œdema about the legs and feet; yet even then the end may not be imminent. But on this part of the subject the reader is referred to the section on diseases of the myocardium (p. 885).

Soldier's heart.—I venture to give this name to a disease well known to physicians in the army, not by any means with the intention of confining the class of cases now to be considered to the soldier, but to indicate a state of heart which is peculiarly apt to occur in him, as its causes are of kinds to which soldiers are more exposed than civilians. Nevertheless if civilians, or men in other services, are exposed to like influences, they also will be liable to "soldier's heart." Our attention was first drawn to this condition by Brg.-Sur. Lieut.-Col. Myers more than thirty years ago; and his most recent views on the subject will be found in Quain's *Dictionary* under the head "Exercise." In the United States the subject was first studied by Dr. Da Costa. Many cases of the kind, occurring in civil life, come under the notice of the general physician, so that the condition is now well known. The degrees of the malady range between the transient disorder of the heart seen in any youth who in a somewhat too reckless pursuit of exercise may be disturbed with some palpitation and dyspnœa for a few days or weeks only, and a persistent disease of an incurable severity. The former transient cases fall under the head of "Irritable Heart," in the chapter on "Functional Disorders of the Heart" (p. 807); the latter fall into the present section on "Strain of the Heart." The differences are indeed no more than of degree; but in comparing the extremer cases we find a difference of degree amounting to a difference in kind.

In Quain's *Dictionary* Myers says: "The young soldier of light frame, with irritable, palpitating heart, who has broken down in his preliminary training, is a marked and good example of the early injurious effect of overstrain of the heart, under the impediments, caused by tight

clothing and accoutrements, to the free expansion of the chest. Whe
rest he feels perfectly well, and has little or no throbbing in the ch
So soon, however, as he puts on his tunic and accoutrements, and beg
his drill, throbbing occurs with more or less violence, accompanied w
a feeling of oppression and with difficulty of breathing, and this be
followed by a sensation of faintness, sickness, or dizziness, he has to
out of the ranks. At first the condition is one purely of functional
turbance which, though rendering him unfit for the duties of a soldi
does not interfere with his gaining his livelihood as a civilian." In
cussing the late Dr. Morgan's evidence of the safety of athletic pursu
afforded by the experience of University oars during the years 1820-18
Myers properly warns us that these were men picked for their large fram
full chests, and exceptional strength. On the other hand, from a la
experience of University men, I must say that considering their "viol
and unguided efforts to achieve success" and their "ill-regulated emu
tion," the ill effects are surprisingly small. Many are the "irrita
hearts" (p. 821), but permanently or gravely injured hearts are f
or none. On the other hand, I agree with Myers in his admoniti
to "men who have settled down into the real business of life who, duri
their nominal periods of rest from their daily labours, undertake viol
exercises without any preliminary training, and thus throw such
unexpected strain on the heart and blood-vessels that instead of m
functional disturbance, as in early life, they sow the seeds of orga
disease." Although I am tempted to minimise the allegations of seri
harm to the emulous young men (for among them there is now a sort
natural selection, the weaklier taking to girls' games, such as lawn tenn
hockey, cycling, and the like), yet I cannot enforce too strongly
warning to older men who are not in the casual training in which
healthy youths are constantly, if more or less unsystematically, engag
Bear-fighting among themselves, running and shouting with the games
others when not themselves at work, bounding up and down long fligh
of stairs, scampering, always a minute too late, to lecture or chapel, th
are always more or less in training, and, being well and plainly fed a
devoid of care, they bear what the elder brother cannot bear, who g
to his work in a stuffy office by underground rail, loafs to his club in
hansom, dawdles at dinner-parties and At Homes, takes his exerc
vicariously by watching the games of others, and spends the lave of
time with his feet on the chimney-piece with the eternal cigarette in
mouth. This overfed and self-indulgent person, who is plucky enou
when needs must, is surprised that he goes to pieces when, on his mont
holiday, he competes with mountaineers or sportsmen who are in fit c
dition, and who live sparingly. If this be true of the eldest son, what
the father, who will not be forgotten but, with his nervous system corrod
by drudgery and care, is determined to scorch on his bicycle, or to cli
the Alps with any of them. These forcing kinds of effort it is which
for evil far more than ordinary sports by field and stream, which ne
lead to strain of the heart.

One of our younger graduates, Dr. M'Carthy, has recently taken up this matter of soldier's heart in an exercise for his degree. He obtained his materials at Netley. After stating that the modern valise equipment is less injurious to the young soldier than the old knapsack, which by its cross belts constricted the chest, he adds that the malady is still common enough nevertheless. He was able in a short time to collect twenty cases, and also to examine the first batch of twenty soldiers invalided from the campaign on the Indian frontier, and of these again five were found to be patients of this class, though not included in his series.

In dealing with his twenty cases, M'Carthy took out in each the age, total service, the trade of the recruit before enlistment, the habits as to tobacco and alcohol, the climates of foreign service, and the infectious and other diseases which he might have undergone. Fourteen of the men were in infantry regiments, three in the Royal Artillery, two in the cavalry, one in the Royal Engineers. At the date of examination two were under the age of twenty-one; fourteen were between twenty-one and twenty-five; four were of twenty-five years and over. "Taking the statements of the men as true," the average amount of beer consumed daily was from three to four pints. Other alcoholic drinks were taken but occasionally. The average amount of tobacco was three to four ounces a week, the tobacco being generally twist or plug. Twelve had suffered from syphilis; fifteen from malarial and other tropical fevers; two only from rheumatism of any kind. Some of the men figured, of course, in more than one of these categories.

"The patients state that while not exerting themselves they feel quite well and free from any shortness of breath; but as soon as they begin to march they are troubled at once with a throbbing sensation in the chest; and with this there is difficulty of breathing, followed in some cases by faintness or giddiness. Rest may relieve for a time, but in most cases all the trouble returns shortly after returning to duty."

On the other hand, many men (not in the above list) have the disease, yet state that it has never been of any inconvenience to them whatever. "In fact, many cases of disordered heart have been detected quite by accident while going through the usual routine examination, when soldiers come into hospital for other complaints, especially malarial fevers."

To take the *symptoms* in detail:—Cardiac pain was present in seventeen cases, dyspnœa in seventeen, giddiness in six, sleeplessness in five, nervousness in seven cases. Three cases were noted in which the men were unaware that there was anything wrong with the heart.

Physical signs.—In fourteen cases the pulse was regular while the patient was at rest, but in some of these it became irregular after slight exertion; in the remaining six it was irregular even when the men were confined to their beds. In twelve cases the pulse during rest was below 100; in six it was between 100 and 115; in two between 115 and 120. The pulse rarely exceeded 120 when the man was at rest, but would always rise very rapidly to 140 or so on his swinging the arms three times round the head. In nearly all the cases the pulse was of abnormally low pressure.

The area of cardiac dulness was increased in fourteen cases; bu some of them the increase was so slight that it was recorded with he tion. In all the cases the impulse was diffused, and in many the was displaced—in two cases between 1 and 1½ inches outside the ni line. Abnormalities of the cardiac sounds were uncommon. In cases the second sound was reduplicated at the base; in five the monary second sound was accentuated; in six the first sound was sh in three, prolonged and booming; in four cases there was a sys murmur at the apex.

Dr. M'Carthy lays stress on the history of malarial fevers in man these men; he reminds us of the evil effect of fevers on the car muscle, and urges that soldiers recovering from these fevers should exempted from drills and other manual work for several weeks a discharge from hospital.

Alcohol is the next cause on which he lays stress; and, as to toba he says that men smoke more in the tropics where they loaf more; that the tobacco is bad and strong. On campaign the rations also often necessarily short, while the labours are excessive. Finally, author urges that tropical heat reduces the value of hæmoglobin in corpuscles of the blood, and leads to anæmia. If in this condition soldier is called upon to do hard muscular work, is badly fed, and may attacked by some fever, the softened and flabby heart muscle yie dilatation occurs, and the man is invalided.

The *prognosis* is not good; in the majority of cases the patients ret to hospital till they are invalided out of the service. The author fo the difficulty which might be expected in tracing the men thus invali However, he obtained records of thirty cases of men discharged from Netley Hospital, and his impression from these returns is that in m cases the soldier's heart ends in valvular disease. It is said in Cambri that influenza is very mischievous in lodging-house keepers, who ca keep their beds and are frequently running upstairs.

As in various parts of this article I have more or less incident referred to cases of this kind as they occur in civil life, I have nothin add to this careful inquiry of Dr. M'Carthy. I have already said the obstinacy of these cases is remarkable. Those which pass the between "irritable heart" and "soldier's heart" rarely end in recov but in permanent dilatation often resulting, sooner or later, in mi insufficiency. I have only to add that these cases are not only unrelie by cardiac gymnastics (Nauheim methods and the like), but are aggrav by such means. Although muscular exertion is the determining, perhaps an indispensable, cause of "soldier's heart," yet it manife depends also upon many contingent conditions.

I cannot conclude this section without a formal opinion, foun on thirty years of close observation of heart stress, that the importanc muscular effort as a factor in cardiac disease has been much exaggera I have shown that in the sound adult organism the effects of phys stress upon the heart are promptly counteracted by equilibrating machinery, and especially by large expansion of muscular and pulmonary areas. Such a statement as that made three years ago by the editor of a leading medical journal, namely, "that the violent strains of hard exercise bode in the end the certainty of premature decrepitude," and that "the heart can only perform a certain total measure of work," so that "whether this be done by a rapid or a slow process determines the length of days in which it is done," seems to me, both on clinical and physiological evidence, to be unjustifiable.

The clinical story of strain in the aortic area of the heart will find its place in a later section.

T. Clifford Allbutt.

N.B.—For references the reader is referred to the list on page 966.

INJURIES BY ELECTRIC CURRENTS OF HIGH PRESSURE

Since electricity has come to be so widely employed, and is being increasingly used as an illuminating agent and for motive power, accidents of varying severity have been frequent. It is desirable, therefore, that we should be cognisant of the effects of high electrical currents upon the human body. We know that there is considerable danger attendant upon the generation of electricity, and we look to the expert electrician to adopt measures to prevent accidents. During the four years ending 1896 twelve deaths occurred in this country from electric shock; and when we add to these the many lesser accidents that frequently occur, we recognise the need for careful precaution wherever electricity is being generated and distributed. Many of the accidents have been due to inadvertent contact with exposed parts of highly charged metal not properly insulated. The consequences of the current thus passed through the body vary with the amount of current entering, the insulated position of the individual at the time, and the kind of contact. Such conditions, for example, as standing on wet earth, the wearing of damp boots, and a moist skin tend to increase the effects of an electrical current. The danger, therefore, is not one simply of high potentiality of current, but of current plus the conditions under which it has been received. The word voltage used in this article is synonymous with "pressure" as used by the Board of Trade, and with the "electromotive force" of the text-books.

It is difficult to say what voltage is fatal to man. Speaking in terms of voltage Dr. W. S. Hedley says that 1000 to 2000 volts will kill. In America, where electricity was adopted as the official means of destroying criminals, a current of 1500 volts has been regarded as capable of effects are produced at the moment of the entrance into and exit of currents from the body, and that these, therefore, are periods of danger. Hedley, in supporting the opinion that the quantity of electricity passed determines the amount of electrolytic action and physiological effect, considers that more pain is felt the higher the electromotive force, even when the current is the same. One element entering into the causation of pain is the local action of the accumulated products at the point of contact consequent upon electrolytic decompositions, and the relative resistances between the electrodes and the different layers of the skin. The individual through whose body there is passing an electric current of not too high potentiality generally experiences pain, but some of this must be due to the extreme contraction of his muscles quite apart from the influence of any products of electrolysis. If there be no immediate loss of consciousness, terror may cause him to faint. The memory of this plays no small part in the subsequent development of nervous symptoms. Once liberated, the patient, as a rule, is soon well again, but there are instances on record where for many months after exposure to the current there was complaint of ill-defined pains and headache which recurred with electrically disturbed conditions of the atmosphere, and of a form of persistent nervousness which was rather the result of the mental than of physical shock.

Another consequence of the exposure to high electric currents is burning. That portion of the surface of the body which has accidentally been brought into contact with the charged metal may become black and charred, the peculiarity of such a wound being that it is sometimes deep and apt to slough, and that while the burned part is insensitive to pain the surrounding tissues are extremely sensitive. If the skin at the time of contact was moist so much more severe is the burning. If a current sufficient to produce this severe local burning pass through the body, fatal results are the more probable; but if the current merely pass locally, as from the hand to the wrist, for instance, the damage will probably be local only.

When, therefore, the pressure has been high, the contact good, and conditions of resistance slight, the patient may at once be rendered unconscious, or be suddenly killed. Thus stricken by a powerful current a man suddenly falls, or he is thrown a distance of several feet before falling. A peculiar cry is involuntarily uttered, especially when the contact is broken, which, in electrical generating stations, for example, at once attracts workmen to the spot where their comrade is lying, pale, or slightly cyanosed and pulseless, apparently dead, and with mucus escaping from his mouth and nose; now and then a feeble and gasping respiration is observed, but he lies helpless, his pupils keep dilating, and unless artificial respiration is at once resorted to, and sometimes even then, death is inevitable. There is something appalling in the extreme suddenness and severity of the shock in these cases, towards which the unexpectedness of the accident possibly contributes largely.

Cause of death.—In conjunction with Dr. R. A. Bolam I undertook a series of experiments in the Physiological Laboratory of the Newcas College of Medicine upon anæsthetised dogs, with the view of ascertaini the cause of death by electric shock, and of testing the means of resusci tion (7). Two opinions are held by the profession: (i.) that death und such circumstances is due to respiratory arrest; (ii.) that it is conseque upon sudden cessation of the heart's beat. By placing dogs under t influence of ether we were able to take a tracing of the arterial pressu and respiratory movements, and thereby to record the effects of hi electric currents passed into the body. Immediately on making conta the animal is thrown into an attitude of opisthotonos, its muscles becon extremely rigid, and as a consequence the lever recording respirato movement is suddenly and violently thrown up, whilst the other, whi traces the arterial pressure and heart-beats, suddenly rises owing general arterial constriction, and falling shortly afterwards oscillates rapidl but within a narrower range. On breaking the current the respiratic becomes deeper and quicker than before the shock, and in the course a few seconds the breathing and the beat of the heart return to tl normal. When the current proved fatal there were the same initi respiratory and general muscular spasm, and a sudden rise of arteri pressure followed by an immediate fall; one or two quivering oscillatior of the lever mark the arterial tracing, and then all at once a further an complete fall of the lever follows, indicating that the heart has ceasе to beat. Respiration deep and spontaneous may continue for sever seconds, or even for a few minutes after the heart has ceased to bea The experiments invariably showed that in electric shock the deat was cardiac and not respiratory. Other steps were taken to confirm th opinion, notably by listening to the heart of the animal with the steth scope as the current entered. If the current were insufficient to kill th dog the heart's beat was momentarily delayed and then quickened, th cardiac sounds being well maintained; but when, on the other hand, current of higher potentiality was employed, the sounds of the heart woul cease, immediately or very shortly after contact. Respiration deep an rhythmic might continue, but if no treatment were adopted the cardi sounds would not return; increasing pallor would gradually steal ov the whole surface of the body, the pupils meanwhile dilating, and muc being forcibly driven from mouth and nares. By exposing the heart other anæsthetised dogs, and inserting a canula into the trachea so as carry on artificial respiration, Bolam and myself had ocular demonstratio that it was the heart which was primarily arrested in death from electr shock, and not the breathing. Dr. A. M. Bleile (3), Professor of Physi logy, Ohio State University, in a paper read before the American Institut of Electrical Engineers, Niagara Falls, N.Y., June 27th, 1895, states th "death in electric shock is really due to the fact that the current pr duces a contraction of the arteries through an influence on the nervou system, and that this constriction of the arteries throws in such mechanical impediment to the flow of the blood as the heart is unable overcome, and that where drugs are given to counteract this effect, muc larger doses of electricity can be borne." As to the constricted state of the arteries, we ourselves found, with Bleile, that if nitrite of amyl were inhaled by an animal before the electrical experiment much stronger currents could be borne. My results, then, and those of Dr. Lewis Jones likewise, are opposed to those of D'Arsonval, who attributes death to asphyxia.

Morbid anatomy.—There is usually well-marked rigidity of muscles. The skin may or may not show any signs of burning or of eschars; it may be pale or livid. The abdominal viscera and large veins are usually deeply congested. The heart is usually flaccid: sometimes the right side is flaccid while the left is hard and tense. The right auricle and ventricle are considerably distended and are filled with dark fluid blood; the left auricle is generally in moderate distension and contains fluid blood, whilst the left ventricle is firm and almost empty. The lungs present nothing abnormal; they may be slightly congested or at places show ecchymoses, particularly if artificial respiration has been attempted. The brain and spinal cord are congested, but are otherwise normal. I have seen it stated at a coroner's inquest, a diagnosis was based upon the assertion, and the corresponding verdict of the jury returned, that in death from electricity the blood is fluid and not coagulated after death. This is too sweeping a statement, and not quite correct. In most cases, it is true, the blood is found fluid after death, but in some of our experiments we found coagula in the right side of the heart, and occasionally some of the large veins were blocked by dense dark clot—particularly when the autopsy was made twenty-four to thirty hours after death. It is maintained that on spectroscopic examination the oxyhæmoglobin of the blood is reduced. If a strong solution of blood is examined, only one broad band may be observed in the spectrum, and it appears at first sight as if this were due to reduced hæmoglobin; but where the spectrum is very carefully scrutinised, and particularly, too, when the solution of blood is further weakened by the addition of water, two distinct bands of oxyhæmoglobin can be clearly discerned. It would appear, therefore, that the blood contains both oxyhæmoglobin and reduced hæmoglobin. The blood on microscopical examination shows very marked crenation of its coloured corpuscles. The pupils were invariably found widely dilated immediately after death.

Treatment.—Persons who have received only a slight shock and who have not been rendered unconscious require no special treatment. The effects almost immediately pass away, and should any nervous symptoms remain they must be treated on general principles. For any burns or wounds ordinary surgical remedies will avail. It is to the treatment of persons who have been exposed to high electrical currents, and who are apparently dead, that the following remarks apply. D'Arsonval (1), believing the mode of death to be akin to asphyxia, recommended artificial respiration, and of all modes of treatment, quite irrespective of whether the death has proceeded from failure of the respiratory centre or of the heart, I know of no treatment more likely to be beneficial than artificial respiration, systematically carried out by Sylvester's method, and c tinued for half an hour or longer. Bolam and myself have tw succeeded in resuscitating a dog whose heart had ceased beating, once thirteen minutes, and on the second occasion for eight. The heart, wh was exposed to view, had become rapidly distended so as to bulge out pericardium, and had become perfectly motionless after having pas through a stage of fibrillary tremor; but by persisting in artifi respiration, aided by the occasional spontaneous inspirations which fr time to time occurred, and the rhythmic traction of the tongue, contents of the right side of the heart were gradually aspirated into a through the lungs, auricular beats were re-established, at first irregula and feebly; gradually, however, they became stronger and passed o into the ventricle, so that after thirteen minutes, during which the he was apparently irresponsive, we had the satisfaction of seeing the norn beat of the organ restored, the pulmonary and systemic circulation established, and life return. Too often, however, the sufferer is kill outright. Rescuers on approaching the injured must beware lest t current be not broken.

THOMAS OLIVER.

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T. O.

ENDOCARDITIS

I. ACUTE ENDOCARDITIS

Definition and Classification.—By endocarditis we mean inflammati of the endocardium or lining membrane of the heart. The infla mation affects principally and often exclusively the valve segments the endocardium (valvular endocarditis), but other parts of the en cardium may be affected also (mural endocarditis). Both clinically a pathologically we distinguish between acute and chronic endocardi The acute form is again divided into benign or simple and malignant infective endocarditis. Of the chronic form, likewise, we distinguish t kinds,—one which is the result of acute endocarditis, and the other t retractile, fibroid, or sclerotic form, which results from arterio-sclerosis atheroma.

In this article we shall consider acute endocarditis; chronic en carditis, giving rise to the majority of so-called valvular affections of the heart, will be dealt with hereafter.

Acute endocarditis.—In the article on Infective Endocarditis I have already considered the difficulty of separating simple from infective endocarditis. In both forms micro-organisms have been found in the affected valves, though only in the infective form do they play an essential part, so far as symptoms are concerned. While the two kinds have many features in common, in others they differ; and as the difference is often essential we follow the custom and consider the two kinds separately.

ACUTE SIMPLE ENDOCARDITIS.—(SYN.: *Benign, Papillary, Verrucose, Rheumatic Endocarditis.*)

Causation.—By far the largest number of cases occur with (*a*) *acute rheumatic arthritis*, hence by some authors the name acute rheumatic endocarditis is given to the disease. Its frequency in acute rheumatism is differently estimated by different authors; and this is readily to be understood, for in many cases the symptoms of endocarditis may be so slight as to escape detection; or again persons recovering from acute rheumatic arthritis may show signs which simulate those of endocarditis, and yet are due only to some functional derangement of the heart. The most trustworthy observations on this subject are those in which a large number of cases of rheumatic arthritis have been kept under observation, and their after-history watched for some time. Sibson analysed 325 cases of acute articular rheumatism which he observed during fifteen years at St. Mary's Hospital, and found that in 79 there was no endocarditis; in 63 endocarditis was threatened; in 13 endocarditis was probable; in 107 endocarditis was present without pericarditis; in 54 there was endo-pericarditis; in 6 there was pericarditis without endocarditis; in 3 there was pericarditis with doubtful endocarditis.

The proportion given by other observers is somewhat less: the mean of the numbers given by older and more recent writers amounts to 20-23 per cent.

Of other noteworthy facts which have been made out regarding the relation of rheumatism and endocarditis we may note:—

(i.) That, in connection with rheumatism, endocarditis occurs more frequently in children than in adults. Dr. C. West estimated its incidence at 61·3 per cent; Fuller about 66 per cent; and some authors, such as Cadet and Gassicourt, give as high a percentage as 80 per cent.

(ii.) The first attack of acute rheumatism is more often followed by endocarditis than the subsequent attacks.

(iii.) Endocarditis may accompany mild as well as severe attacks of acute rheumatism. Sibson (*loc. cit.* p. 199) states the more severe the rheumatic attack the greater the tendency to endocardial inflammation; but this is not the opinion of other observers, and in children especially we see mild attacks of rheumatism followed by endocarditis. [*Vide* art. "Acute Rheumatism in Childhood," vol. iii. p. 42.]

(iv.) The physical signs of endocarditis usually appear early in attack of rheumatism. Sibson in about one-fourth of his cases not the presence of a systolic bruit, which he looked upon as characteristi endocarditis, at the end of the first week of the rheumatic fever, an two-thirds at the end of the second week. Sometimes, however, signs of endocarditis appear much later, though probably in many of t cases the endocardial affection had existed some time before it gave to physical signs. The endocardial affection may precede the rheum attack by several days.

(v.) Rheumatic fever, or acute polyarthritis, is the disease above others accompanied by endocarditis; but occasionally endocarditis follow monarticular rheumatism and chronic rheumatism. Gonorrh rheumatism stands in close relation to infective endocarditis, though benign form may follow definite attacks of gonorrheal rheumatism.

(vi.) The endocarditis dependent on rheumatism most frequently aff the mitral valve; the aortic valve less frequently, and the right sid the heart in very exceptional cases only.

(vii.) The pathogenesis of what we may call the meta-arthritic en carditis cannot be determined as long as our views on rheumatism ar indefinite as they are at present. The endocarditis cannot be looked u either as a mere complication or as a sequel of rheumatism; it is integral part of the disease. As most pathologists look upon rheum arthritis as an infective and most likely a microbic disease, the poison which chiefly attacks fibrous structures, the endocarditis may be regar as a localisation of the rheumatic poison in the fibrous tissue of the val of the heart. In some few cases the same micro-organisms have b found both in the effusion of the inflamed joint and in the inflammat deposit of the cardiac valves. As in other microbic affections, so proba here, the lesions are due to some toxic product of the microbe circulat in the blood, as is the case in other infectious diseases; to wit, diphthe cholera, and epidemic influenza; and if so, the absence of mic organisms from the deposits is quite intelligible. As the opportunity examining the valve often does not arrive until the endocarditis become chronic, the absence of all micro-organisms, even if the disease microbic, is not astonishing; for this negative condition occurs in cert other diseases which are undoubtedly microbic.

(b) *Chorea.*—Endocarditis is frequently met with in persons have had chorea; and in fatal cases of chorea inflammatory deposits the valves are almost invariably found. Thus Sturges (24) collec statistics of 80 fatal cases, and in only 5 of these were the heart val normal. Reymond's figures bear out the same rule. As regards frequency of endocarditis in chorea authors differ considerably; and, the endocarditis may not reveal itself till years after, the exact p portion is not easily made out. In many cases of chorea a murmur m be due to functional disturbance and not to endocarditis; or, on other hand, as seen in some fatal cases, endocarditis may be present a give rise to no physical signs. Osler states that of 554 cases chorea, at the Infirmary for Diseases of the Nervous System, 170 presented heart murmurs; of these, in 149 the murmur was apical, in 21 basic. Of 449 cases reported to the Committee on Collective Investigation of the British Medical Association, 113 had heart murmurs; how many of these were functional and how many organic it is impossible to estimate: a basic murmur is heard much oftener in purely functional cases, yet an apex bruit may be present from various causes without the existence of endocarditis. More trustworthy results are obtained if the subsequent history of persons having had chorea is taken, an estimate which has been made by several observers. Dr. Stephen Mackenzie examined 33 patients at periods varying from one to five years after the attack of chorea, and noted signs of undoubted heart disease in 60·6 per cent; Donkin in 40 per cent: Osler out of 140 cases found the heart normal in 51; in 17 there was disturbance which might reasonably be looked upon as functional, and in 72 cases ($51\frac{3}{7}$ per cent) there were signs of organic heart lesion: it may be noted that only in 25 of these 72 cases was there a history of acute arthritis.

Nothing more definite can be made out concerning the relation of chorea to endocarditis. So many cases of chorea show signs of acute rheumatism during the course of the attack, or are followed by an arthritic affection having all the characters of acute rheumatism, that the endocarditis has been regarded as a manifestation of the rheumatism only; yet, as will be seen from the figures given above, in many cases where heart murmurs were noted there was no history of acute arthritis; and this is also noticed in fatal cases of chorea, in which endocarditis is almost invariably found. The report of the Collective Investigation Committee of the British Medical Association gives of a total of 439 cases of chorea 97 with a rheumatic history (about 22 per cent). Statistics on this subject, however, are not of great use, as the joint pains occurring in chorea are not always due to rheumatism; and, especially in the severe cases, are probably due to a septic condition. We must note, however, that in children the joint affections in rheumatism may be very slight. I cannot therefore agree with Roger that chorea, rheumatism, and endocarditis are three terms of one and the same pathological series; though undoubtedly in a good many cases endocarditis and rheumatic arthritis, with other signs of a rheumatic diathesis (tonsillitis, subcutaneous nodules, erythema, profuse acid perspiration, etc.), complicate chorea. As yet there is no proof or evidence that chorea is due to minute cerebral embolisms of microbic nature; moreover, the form of endocarditis we notice in chorea is—in by far the largest number of cases—of the benign or verrucose and not of the malignant nature, in which latter the microbe plays the important part. We can but surmise that chorea, by weakening the system, and exercising some deleterious effect on the heart valve, acts only as a predisposing agency.

(*c*) Acute endocarditis may be associated with the acute *zymotic fevers*. Among these scarlet fever occupies the first place. Often we have, before the occurrence of the endocarditis, pains and slight swelling of

a joint, which are apparently rheumatic in nature; thus here
the rheumatic poison is the cause of the endocarditis. Some au
however, look upon the arthritic symptoms as an outcome of the s
fever toxin, and upon the endocarditis as the result of the action
same toxin on the endocardium. In the other acute fevers, su
typhoid, measles, small-pox, diphtheria or malaria, endocarditis is a
rare complication. Pneumonia is more often associated with inf
endocarditis; and the same is the case with erysipelas, with puer
and septic diseases generally, and with gonorrhœa.

(*d*) In cases of *acute and chronic tuberculosis* we meet with endoca
occasionally. Are we to look upon such cases as belonging to the inf
type of endocarditis, or do they belong to the benign form, the tul
bacillus acting as a remoter cause? It must be noted that in a few
the tubercle bacillus has been found (2) in the valve deposits; and in
cases of acute miliary tuberculosis vegetations on the heart valv
recent origin have been observed. I remember in a child, who died
general acute tuberculosis, that the pericardium was found studde
over with miliary tubercle; and the mitral valve showed deposits
proved to be masses of fibrin and leucocytes, and contained tul
bacilli. In this, and probably in a good many other cases, the endoca
is of a specific nature, and therefore, as part of the general disease, be
to infective endocarditis; how far, however, this applies to all cases
valve deposits are found in persons who have died of tuberculosis
only be settled by a microscopic examination of these masses. Ev
the old view that phthisis and heart disease are antagonistic be
strictly true, yet it is rare to meet with either acute rheumatic art
or valvular affections of the heart in persons suffering from phthisis.

(*e*) *Syphilis* attacks the myocardium and the endocardium; in
former it causes endo- and periarteritis with tracts of fibrous tiss
the midst of the myocardium, or it may lead to granular deposits.
the latter case valvular disease may result from arterio-sclerosis, of
syphilis is one of the remoter causes; that acute endocarditis is eve
to the syphilitic virus is very doubtful. Chronic endocarditis of syph
nature does occur, but is a very rare occurrence (8).

(*f*) Of other causes of chronic endocarditis we may mention
and Bright's disease.

Gout.—Several cases are on record (6, 3, 9) in which endoca
processes showing the presence of urate of sodium crystals were fou
persons affected with gout.

In *Bright's disease* we often find chronic valvular heart affection
arterio-sclerosis, yet occasionally it may be associated with acute
carditis (15, 21, 7).

(*g*) *Traumatism.*—Several cases have been recorded, amongst o
by Clifford Allbutt and Litten, where all the signs of endoca
followed a blow or fall on the chest. Litten tabulated the rec
cases and added two more. The endocarditis is most likely due
rupture or injury to the valve; how readily endocarditis is set u

such an injury is proved by the experiments of Rosenbach and others, who, on injuring the valve in animals by introducing a fine wire through the carotid, noticed distinct endocarditis to follow after a few days. Roy and Adami, on lightly ligaturing the aorta, and thus increasing the blood-pressure, produced œdema of the valves and cell exudation.

That infective endocarditis may be occasioned by an injury I have already stated in my article in vol. i. p. 632. The cases cited by Allbutt and Litten were cases of benign endocarditis, some affecting the mitral valve and producing stenosis, and others the aortic valve; in some of the cases, however, the connection between the traumatism and the endocarditis was not satisfactorily established.

(*h*) Endocarditis without any apparent cause, and occurring as an idiopathic disease, has been described by some authors. Such an occurrence is quite possible, yet it must be extremely rare; and probably the endocarditis is in such cases the outcome of acute rheumatism, for, as often happens in children, the joint affection may be quite insignificant.

Other etiological factors relate chiefly to *age*. It appears that endocarditis occurs most frequently between the ages of 15 and 40; it is rare in old people, in whom valvular lesions are mostly due to an atheromatous process; it is not rare in children, as already observed by West, who noticed it 71 times in 122 cases of heart disease (see also on the subject Dr. Cheadle's article in vol. ii. of this *System*, p. 42). In very young children, however, the affection is rare; in them pericarditis is more often found than endocarditis.

Fœtal endocarditis is by no means a rare affection. It may occur with or without congenital anomalies of the heart. As is well known, the right side of the heart is generally affected in the fœtus; but according to the observations of Rauchfuss stenosis of the aorta occurs as frequently as stenosis of the pulmonary artery; and he comes to the conclusion that when there is no congenital malformation of the heart, the left side is as frequently affected as the right. Apart, then, from the anomalies which predispose to right-sided endocarditis, other factors are in play which determine the frequency of right-sided endocarditis, as compared with its rarity in extra-uterine life. Such factors are the thickness of the right ventricle, the increased pressure to which it is exposed, and the absence of pulmonary respiration, which causes such a difference between the blood of the right and left sides of the heart after birth; especially as regards the amount of oxygen. Klebs, who was one of the first to attribute all forms of endocarditis to micro-organisms, gives another explanation; namely, the direct infection of the right side of the heart through the blood coming from the placenta. Recent observations have shown that micro-organisms do not readily pass through the placenta; but, if the micro-organisms do not pass, the toxic substances produced by them may do so, and thus give rise to inflammatory deposits. Fœtal endocarditis cannot well be recognised before birth, and may be

undetected for years after birth. The fœtal right-sided endocarditis
principally the pulmonary valves—often when there is already obstru
or stricture; occasionally the tricuspid valve only: similarly, left
fœtal endocarditis more frequently affects the aortic valve with or wi
contraction of the lumen of the aorta, and the mitral valve only occasio

Finally, endocarditis may be secondary, being an extension
affection either of the myocardium or of the aorta.

Pathological anatomy.—Endocarditis affects principally the
of the heart, hence the name valvulitis; and, except in the intra-u
form, it is almost always confined to the valves of the left side:
again it affects the mitral more frequently than the aortic valve (th
cuspid valve, however, is occasionally also affected in combination
stenosis of the mitral valve). Of the mitral valve it affects the aur
surface, and here again principally the portions of the valve whic
in close apposition when the valve closes; when it affects the aortic va
is found on the ventricular surface round the corpora Arantii. Th
left side is much more often affected than the right side is due to se
causes, but principally to the higher blood-pressure and the differen
the oxygenation of the blood: the first factor leads more easil
abrasion of the endocardium, and other changes favouring the depos
inflammatory material or thrombi, and by the latter the action of the m
organisms is greatly favoured. That the mitral valve is more frequ
affected than the aortic may be due, as Sibson (*loc. cit.* p. 458) po
out, to the fact that the mitral flaps press against each other
the valve is shut with much greater tension and force than
cusps of the aortic valve. To the combined agency of a finer marg
contact, greater pressure of blood, and the muscular force and tend
traction proper to the valve, another fact may be added, namely,
absence of vessels in the aortic and pulmonary valves (Langer, C
a condition which protects them, at any rate, against the invasi
microbes by embolism. The endocarditis is localised chiefly at the
of the valves indicated, because at these spots we have the gre
pressure and stress.

It must be noted, however, that other parts of the endocar
especially the chordæ tendineæ, are implicated in the process.
endocarditis affecting chiefly other portions of the endocardium, t
exclusion of the valve, has been described by Neuwerck; it is mo
less chronic, and leads not only to superficial cicatrices, but also to
endocardial and myocardial inflammation (Rosenbach).

Appearance of the affected valves.—In the early stage, which we
but rarely the opportunity of seeing except perhaps in some case of
chorea, the endocardium at the affected parts is slightly swollen, a
the rosy tint of increased vascularity. At a more advanced stag
notice little pedunculated vegetations, forming a string or garlan
small beads, on the auricular surface of the mitral and ventri
surfaces of the aortic valves; not on the free edges of the valves, b
some slight distance from the border, corresponding to the lines o

maximum contact of the valve segments when the valve is closed. When the chordæ tendineæ are involved, the endocardium covering appears opaque, and slightly raised; and in rare cases may be the seat of small vegetations.

The further progress of these vegetations varies: in rare cases they may be completely absorbed; in other cases the vegetations increase in size and in extent, forming large fungating masses which may extend to the chordæ tendineæ, and cause a serious obstacle to the free circulation of the blood. In most cases the inflammatory deposits undergo fibrous change as in inflammations elsewhere; and these sometimes also affect the chordæ tendineæ, and even the papillary muscles, as seen in some of the chronic valvular affections; or some of the cusps may become adherent to each other or to the walls of the heart. The fibrous and contracted valve segments assume an almost cartilaginous appearance, and, being deficient in blood, may give rise to degeneration of the valves; notably to calcareous degeneration and the formation of necrotic ulcers.

Histologically the affection shows changes of an inflammatory nature in the endocardium, and deposit of fibrin in the form of thrombi, both white and mixed, from the blood. Sections of a small vegetation in a very early stage show proliferation of the endothelial layer, increase of the branched cells in the subendothelial tissue and infiltration of the layers of the endocardium with leucocytes, fibrin and serum between the trabeculæ, and a deposit of fibrin on the free surface of the endothelium; this is deposited from the blood, and may be granular or sometimes fibrillar. When the process has lasted some time this embryonic tissue is changed into fully-formed fibrous tissue; and after a time calcareous particles may be deposited in the newly-formed structures, and the chordæ tendineæ and papillary muscles then show the changes to be described under chronic endocarditis. In the severe cases the myocardium shows indurative changes, which may be looked upon as due to an extension of the inflammatory process; the fibrous septa and the lymphatic spaces being chiefly involved: even the muscular fibres may show changes partly due to compression, and partly to myocarditis.

An examination for micro-organisms may reveal in some of the acute cases the presence of staphylococci, or streptococci, or diplococci (12); when the affection has become chronic, microbes are rarely found. The organisms are found in larger numbers in the superficial layer of the deposits, and but sparingly in the deeper.

From acute endocarditis must be distinguished—

(*a*) Patches of red coloration of the valves, which are sometimes seen in persons who have died of an acute infectious disease. These patches are simply due to blood imbibition.

(*b*) Certain deposits on the auriculo-ventricular valves, which are the remains of an embryonic condition of the valves, as pointed out by Bernays; and in these Luschka has demonstrated pigment particles due to old hæmorrhages (Rosenbach, *loc. cit.* p. 156).

(*c*) A form of endocarditis probably due to sudden disturbance of intra-cardiac pressure, whereby the endocardium is injured. W here small, close, hard vegetations, firmly fixed and without a fibrin (Dickinson).

Symptoms.—Acute endocarditis is sometimes easy, at other tim difficult to recognise; and at times we find after death the charac lesions on the heart valves without there having been any sympt endocarditis during life.

The subjective symptoms vary considerably with the age patients, the primary diseases, and the presence of complications s pericarditis, and the effects of any previous attack of endocarditis. physical signs may sometimes be absent or appear only when th process has passed into a chronic state; and they are sometimes d to distinguish from those of a functional disturbance of the hear some cases the symptoms are absent, and it is only perhaps wh patient is brought under our notice in an attack of hemiplegia by em that endocarditis may be detected.

If we take the acute rheumatic as the most common form of carditis, we find in many cases no subjective symptoms to lead suspect an endocardial affection; the febrile symptoms, such as te ture, pulse, perspiration, do not differ from those in cases of rhe fever without endocarditis; and it is by the physical examination heart only that the existence of endocarditis is detected. In a group of cases the patient, who has generally been affected wi rheumatic fever for a week or more, has suddenly a rise of tempe without any fresh pains; or he complains of oppression, uneasin pain over the region of the heart and palpitation; the pulse b small and quick, and the heart's action tumultuous: in other ca subacute course, dyspnœa on exertion is the only symptom complai yet physical examination of the chest reveals the existence endocardial murmur. In children, when pericarditis complicates en ditis, which it frequently does (the carditis of Sturges), the sympto more pronounced and fairly characteristic; the breathing, with al dilated, is hurried and laboured, and there is great orthopnœa; th has an anxious look and is somewhat cyanotic, sleep is very disturbed, and there is generally marked delirium. The pulse in cases is very quick, small and compressible, and there may be per vomiting. It must be noticed that in children the joint affect rheumatic arthritis may be so slight as to be easily overlooke art. on "Acute Rheumatism in Children," vol. iv. p. 40]. It may h as in a case I saw recently, that the only noteworthy feature is of temperature with profuse sweating, which may go on for some The daily examination of the heart shows at first nothing abn but in a short time the physical signs of endocarditis present selves. In other cases in children, as in chorea, little general distu is noticed.

Physical signs.—The physical signs are sometimes very marke admit of no other explanation; at other times they are indefinite.

On inspection of the thorax nothing abnormal is noticed, unless there be pericardial effusion; nor do we get any evidence of valvular disease on palpation unless the affection has already existed for some time.

On percussion it is only in exceptional cases that we notice the increase of the area of cardiac dulness due to dilatation of the left ventricle, the right, or of both. An increase in the area of dulness, however, more especially in the transverse diameter, is often noticed in children, and may be due to pericardial effusion; if so, the pulse is feeble, the apex beat is not so well felt as usual, and the area of dulness has the characteristic outline of pericardial effusion.

The most trustworthy and important physical signs of valvular endocarditis are noticed on auscultation. As the mitral valve is most frequently affected, and as the fibrinous deposit is apt to prevent the complete closure of the valve, we get the signs of mitral regurgitation; that is, a systolic murmur heard best at the apex, and conveyed towards the axilla and also towards the sternum. In a good many cases of acute rheumatic endocarditis, under my own observation, which afterwards lapsed into chronic valvular disease, a systolic murmur, soft and blowing in character, was noticed at the apex; but as a rule better heard over the lowest portion of the sternum close to its junction with the left costal cartilages. The appearance of a systolic murmur is preceded for days by an impurity and prolongation of the first heart sound which is in itself suggestive of endocarditis. Prolongation of the first sound is the first whisper of an "approaching murmur" (Sibson, *loc. cit.* p. 493). This is probably due to the soft gelatinous deposit, which alters the first sound while the valves are still smooth and elastic. According to Sibson, we may notice occasionally, besides the mitral bruit, a tricuspid systolic murmur also; but this is not heard at the very beginning of the endocarditis (Sibson, *loc. cit.* p. 242). As regards acute endocarditis in children, Sturges (25) gives as the earliest physical indication: "Tumultuous, quickened, and uneven heart's action and sounds, that are changeful from day to day, especially the first; sounds reduplicated, at and above the apex (not at the base); a temporary tricuspid murmur; marked accent commencing the first sound, whether mitral or tricuspid." Occasionally, however, even in children, a loud systolic murmur may rapidly appear; this is sometimes only heard when the child lies down; in the erect position it becomes fainter and may even disappear.

In acute febrile affections, such as the acute zymotic diseases, and in rheumatic arthritis, a systolic murmur may be heard under conditions other than that of endocarditis; therefore, when we hear such murmur we must not conclude at once that there is endocarditis. The murmur may be due to relaxation or other changes in the heart muscle, or to a change in the blood (hæmic bruit).

Although it is not always easy or even possible to distinguish these conditions, certain signs will help us. The pulse in myocardial affections is often quick, small, and irregular; and there is marked dyspnœa and vertigo. The hæmic murmur is noticed when there is well-n anæmia; it is heard not only over the mitral, but often also ov pulmonary and aortic areas, and is accompanied by venous murn the neck, while the pulse may be dicrotic. (According to Sansom, n dicrotism occurs only in the severe cases of endocarditis.) Besid mitral murmur, especially if the heart's muscle is weak and early dila of the right ventricle comes on, we may note reduplication of the s sound, accentuation of the second pulmonary sound, and sometim a systolic bruit over the tricuspid valve.

If the endocarditis affects the aortic valves we may find no s physical signs if the vegetations are very small; at other tim get evidence of aortic regurgitation, a diastolic bruit heard best at sternum; and, if there be much regurgitation, we get other indic of aortic incompetence.

The physical signs denoting stenosis of either mitral or aortic are very rarely to be noticed; as the narrowing results from a contr of the valves which generally takes place as the endocarditis be chronic. Dr. Sansom states that in some cases he has observe duplication of one or other of the heart sounds as an early sign of carditis; and in these cases the endocarditis was followed by st rather than by regurgitation (22).

In the rare cases of right-sided endocarditis we have the sig tricuspid or pulmonary regurgitation.

Complications.—Leaving out of consideration the rare cases—v however, mostly belong to infective endocarditis—where there is ru of the inflamed valve or rupture of chordæ tendineæ, the most fre complications of endocarditis are *pericarditis* and *myocarditis.* Sibs 161 cases of acute endocarditis noticed that pericarditis was prese 34 cases, and in children the proportion is even larger. Changes i myocardium varying very much in degree have, according to r observations, been found so often that they too must be looked upon as mon; though they do not often give rise to symptoms which lead to recognition. Yet we may suspect their occurrence if the heart's a becomes weaker, or if there are physical signs of an acute dilatation heart; the pulse becomes quicker, weaker, and often irregular, the beat weaker, and the murmur less distinct; there is also marked dys and the patient complains of tightness and oppression, and occasic of pain and palpitation: these signs are often followed by vertigo, deli and cold, clammy perspiration. Cheyne-Stokes breathing has some been noticed towards the end, and death takes place either from sy or pulmonary congestion and œdema.

Pleurisy and *pneumonia* are occasional complications. The re of pneumonia to infective endocarditis has already been noticed (p. 633). Rheumatic endocarditis may, however, be associated croupous pneumonia, or it may give rise to embolic pneumonia.

Embolic infarcts occur more frequently in infective endocarditis in chronic valvular diseases, than in the acute rheumatic endoca

In some rare cases (11) the endocarditis propagated to the aorta may produce acute aortitis, a complication which is difficult to diagnose; it is said to produce severe paroxysmal pains behind the sternum with radiation towards the shoulder, dyspnœa, and perhaps diastolic aortic murmur.

Course and termination are most variable; in some cases the symptoms may disappear, and the patient completely recover; in others the patient apparently recovers, but for some time looks very anæmic, and the physical signs never disappear. Or the patient may enjoy excellent health and be not aware that he has any valvular lesion till many years afterwards, when the first symptoms of want of compensation of the heart-defect make themselves felt; the length of time before these symptoms come on depends on many factors, such as the extent of the lesion, the condition of the heart muscle, the occupation of the patient, intercurrent diseases, and so on. In other but fortunately very rare cases, where the valvular lesion is very severe, or the myocardium very much enfeebled, the symptoms denoting failure of compensation (dyspnœa, quick, weak, or irregular pulse, œdema and dropsical effusion) may come on early after the onset of the disease. When pericarditis complicates the endocarditis the patient may pass years without any serious troubles, till the compensation, whether of the valvular defect or of an adherent pericardium, begins to fail.

Death may take place during the acute stage from the presence of complications such as pericardial effusion, myocarditis, pneumonia, embolism, or in some rare cases from hyperpyrexia; or some infective agent may convert the rheumatic into infective endocarditis.

In children, in whom the physical signs are usually well pronounced, and pericarditis often present, all the signs may completely subside, and a restoration to complete health take place; in most cases, however, the child apparently recovers and may enjoy good health for many years in spite of the presence of signs of valvular disease; yet eventually, either without apparent cause or on the appearance of some incidental disease, he manifests the subjective and objective signs of valvular disease. In some few cases belonging to the group called active carditis by Sturges (*loc. cit.* p. 506), death takes place from the associated pericarditis or from pulmonary œdema, embolic pneumonia, or cerebral embolism. The cause of death in some cases of chorea with endocarditis is often very obscure, and not due directly to the endocarditis.

Diagnosis.—From what has been said of the symptoms of acute rheumatic endocarditis, it will be clear that the diagnosis, though easy in some cases, is occasionally impossible; in many cases, indeed, the endocarditis can only be suspected. When no murmur is heard over the precordial region we can only suspect endocarditis when, say in a case of acute rheumatism, the heart sounds become veiled and impure (such changes are best noticed when the heart has been daily examined), and the patient complains of palpitation or oppression, as pointed out above. When a murmur is heard over the region of the heart we have to distinguish

between an exocardial and endocardial murmur, and if endocardial w
it is due to endocarditis.

The exocardial murmur, which is occasionally difficult to discrin
especially in children, is a *pericardial friction sound;* but the cha
the rhythm, the situation, the variability of the murmur, the dir
in which it is propagated, and some other points will help us to distin
pericarditis from endocarditis. Thus in pericarditis a double murn
heard which does not replace the heart sounds, but only obscures
nor is the double murmur synchronous with them: the murmur may
the character of a hard or soft friction sound; it is heard usually
the right ventricle, though it may be audible with less intensity ne
apex; it appears to be superficial, is localised over a small area, i
propagated either to the axilla or along the sternum, and it is va
within short periods of time. Occasionally the rub may be felt whe
hand is placed over the precordial region. If there be much effusio
precordial region may bulge; the area of cardiac dulness increases
has a peculiar pear-shaped outline; the apex beat is raised, disp
towards the left and indistinctly felt; and, on auscultation, tu
breathing may be audible over a small area of the back. In perica
pain and oppression are often noticed.

An exocardial murmur may be due to pleurisy. As a rule th
no difficulty in distinguishing the pleuritic rub from pericardial fr
and from an endocardial murmur, since when the patient is ask
hold his breath it disappears; but occasionally the pleuritic rubs, th
lessened, persist and are rhythmical with the heart's contraction:
rule, however, the rub extends towards the left beyond the limits o
heart, and there is often pleuritic pain.

Another exocardial murmur may sometimes be heard above the
beat towards the left; it is rough, varies in intensity, begins afte
systole, and disappears when the patient sits up and bends for
According to Potain, this murmur is due to the intrusion of a
layer of the lung, close to the heart, into the space before occ
by the base of the heart, as with each ventricular contraction the
is projected forwards and the base retreats slightly from the
wall.

Having eliminated the exocardial murmurs, we have yet to deter
whether the murmur be due to endocarditis (so-called organic murmu
functional; and, if due to endocarditis, whether recent or old, benig
infective. The chief points of distinction between the hæmic murmu
the murmur produced by the dilatation of the heart, and by endocar
have been given above. As a rule there is no difficulty in distingui
recent from old endocarditis; we have to take into account the hi
of the case—whether there have been previous attacks of rheumatis
chorea, or of some of the other diseases followed sometimes by
carditis; or whether the patient has suffered from dyspnœa on exc
or œdema of the feet. The presence of secondary changes in the
due to chronic valvular disease, such as hypertrophy of the left ven

or dilatation of the right ventricle, indicated by accentuation of the second pulmonary sound with signs of venous stagnation (œdema, enlarged liver, albuminuria), are of great help; but we have to bear in mind that dilatation of the right heart may come on occasionally in acute endocarditis, and that a previous attack of rheumatic endocarditis favours the recurrence of such attacks, should the patient suffer again from acute rheumatism; thus we may have an acute endocarditis implanted on an old one.

The discrimination of rheumatic or benign from malignant endocarditis will be considered when treating of the latter disease.

Prognosis in acute endocarditis is sufficiently evident from what has been stated concerning the course and termination of the disease. Death during the acute stage is generally due either to the severity of the primary disease—be this rheumatism, chorea, or an infective fever—or to some complication, such as myocarditis, pericarditis, or pneumonia; in some rare cases symptoms of dilatation of the right side, with venous stasis, shown by dyspnœa, dropsy, irregularity of the heart's action, may come on and lead to death. A very large majority of patients recover from the acute attack, remain well for years, but become the subjects of chronic valvular disease; and this may occur in cases in which the murmur had disappeared for a time; lastly, in some few instances complete and permanent recovery takes place. When an acute endocarditis occurs in persons already affected with valvular disease the prognosis is still more serious; for often the fresh endocarditis is of the infective or malignant kind; or, even without this, the fresh deposit may lead to embolism or, by increasing the weakness of the heart, hasten the downward course of disease.

Treatment.—*Prophylactic treatment.*—As acute endocarditis is associated most frequently with acute rheumatism, our attention must be directed to prevent the occurrence of this malady in persons with a family or personal proclivity to the disease; such persons should wear flannel next to the skin, avoid living in damp houses and in districts where clay forms the subsoil and rheumatism abounds, and avoid as much as possible those sudden changes of temperature which are so apt to produce chills.

If a person is taken with acute rheumatism, can we by speedy and proper treatment prevent the occurrence of endocarditis? This question has been the subject of many discussions, especially since the introduction of the salicylates, which have such a decidedly beneficial effect in acute rheumatism, often causing a speedy disappearance of the symptoms, and cutting short the duration of the disease. There is, however, now abundant evidence that the cardiac affections are not warded off by this treatment [see discussion in the Medical Society of London, 1881]; on the other hand, we cannot say that their frequency has been increased by this now universal treatment of rheumatism. Some maintain that the treatment of acute rheumatism with large doses of alkali, combined with absolute rest in bed, has a more protective effect against the cardiac

complications than the salicylates or salicin; and many now us
combined treatment. So far the prophylactic treatment has ha
little success; yet it is most important that every case of acute rheun
in adults, and still more the various modified and less pronounced
in children [see Dr. Cheadle's article on "Acute Rheumatism in Chil
vol. iii. p. 52], should be treated at once by rest in bed, with cor
repose and appropriate medicine (alkalies and salicylates).

Local treatment.—Venesection, recommended by Bouillaud ar
school, and extensively practised for years, need only be mentioned
historical interest. The application of a few leeches to the prec
region, especially in young and plethoric subjects with a quicl
full pulse, and when there is precordial pain and oppr
indicative of early pericarditis, may be safely recommended fo
relief of these symptoms. The local application of ice, long since r
mended by Friedreich, and extensively practised on the Continen
been warmly advocated by Dr. D. B. Lees, especially when peric
complicates endocarditis; it may be tried also in simple endocardit
reduces the fever, diminishes the frequency of the pulse, calms the
of the heart, and relieves such subjective symptoms as pain and o
sion. It is well to apply flannel next to the skin and the ice-bl
over the flannel; for dry cold is much better borne than wet cold
a rule it is quite tolerable, and indeed comforts the patient. It is c
indicated when there is marked cardiac dilatation with a smal
intermittent pulse; but even when these conditions obtain, it m
cautiously tried for a short time.

Other local remedies used are blisters, sinapisms, and tinctu
iodine. Large blisters have often been recommended as derivative
recently Dr. Caton has spoken favourably of repeated small bliste
have often applied blisters both in endocarditis and in endo-perica
and with relief of some of the subjective symptoms; but I cann
that they have influenced the disease very much. Painting th
cordium with tincture of iodine, repeating this from time to tim
persisting with this for weeks or months, is asserted by some obs
to be attended with good results.

General treatment.—With the appearance of the first sympto
endocarditis some physicians recommend the administration of
doses of alkalies and suspension of the salicylates, which h
depressing effect on the heart; others see no objection to a contin
of the salicylates, unless signs of failure of the heart or of myoca
appear; others again prefer to give salicin, which has a much le
pressing effect. As, however, with this treatment the endocarditis,
once it has shown itself, is rarely completely cured, I have tried from
to time both local and general means to check, if possible, the infla
tion of the endocardium and to minimise the damage done by it.

Tartar emetic, as recommended by Jaccoud, is scarcely ever emp
by English physicians; nor do many of us give mercurial prepar
which were once so highly spoken of both by Graves and by S

except in obedience to special indications. Iodide of potassium has been given at a later stage of endocarditis to hasten the absorption of the deposits.

As essential as the medicinal treatment is the general management and diet of the patient. The patient must be confined to bed for weeks, kept quiet, and all excitement avoided; the diet should be light, but nutritious, and, unless the heart show signs of failure, stimulants had better be avoided altogether.

Other drugs than those given above may be indicated by certain symptoms and under certain conditions. If there be much pain and restlessness small doses of morphine may safely be given. Antipyretics are only indicated when the temperature is high and the pulse very quick. Quinine in fairly large doses (15-20 grains), or phenacetin (gr. 5 to gr. 10), are preferable to antipyrin or sodium salicylate. Digitalis is not required unless the pulse becomes quick and small, or irregular; the tincture of digitalis or digitalin may be given when signs of cardiac failure appear. Besides this drug we may give strychnine, ammonia, brandy, and ether under the above conditions. When there is much dyspnœa and cyanosis inhalations of oxygen will be found useful, especially in children. In cases in which the pulse is quick but full, and in which the heart's action is good, digitalis had better be avoided, as an increase in the force of the heart might lead to a detachment of clots or parts of the vegetations, and thus to embolism.

For the anæmia, which often persists for weeks and months after the acute symptoms have passed off, preparations of iron are given with quinine and arsenic; the latter drug appears, indeed, to have a better effect than the iron preparations. Convalescents from acute endocarditis should be sent for some weeks into the country or to the seaside; a dry, bracing climate being preferred. If there be much subsequent dilatation the Nauheim treatment may be tried, and the other measures recommended for chronic valvular disease [see later articles].

Those cases of rheumatic endocarditis which assume a malignant type, which run a long and protracted course, and in which fever persists, rigors and hæmorrhages appear, and further complications (septic pneumonia, embolic abscesses) arise, require the same treatment as cases of infective endocarditis, to which class indeed they belong.

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II. Infective Endocarditis

Syn.—*Malignant Ulcerative Endocarditis*

I have already (vol. i. p. 626) considered the causation and
geny of this form of endocarditis; it remains now to discuss the
logical anatomy, symptoms, prognosis, and treatment.

Pathological anatomy.—Lesions are found in the heart a
various other organs of the body. Some are primary, and represe
seat of inoculation; others are secondary, but the most import
these are produced by the micro-organisms circulating in the blood.
changes found in the heart vary considerably according to the mi
which produce the disease, the extent of it, its duration, and esp
whether the infective endocarditis affect a healthy heart, or one al
the seat of old endocarditis or sclerosis of the valves. We may have
vegetations, or—and this is the most frequent occurrence—we find a
or less extensive ulceration of necrotic character; or, occasionally
the formation of one or more abscesses. Malignant endocarditis
simple or rheumatic endocarditis, principally affects the valves; and
more frequently the valves of the left side than those of the
though the latter are more liable to be affected than in rheumatic
carditis: thence it may extend to other portions of the endocardium
to the aorta or pulmonary artery. Mural infective endocarditis, in
the valves remain free, is extremely rare.

When there are only vegetations these are generally small
grayish or yellowish in colour; they affect the base as well as the m
of the valve. Such a condition we sometimes see in cases
run a very rapid course; histological examination of the valve r
numerous microbes, embryonic cells, and leucocytes; besides the lay
fibrin. In most cases, however, the vegetations are larger, occasio
pedunculated, and more or less extensively ulcerated; these ma
superficial, not extending deeply into the tissues; they are gr
and often partly covered with fine blood coagula; if the valve yie
the blood-pressure, depressions (aneurysms) may result. Sometime
ulceration may penetrate deeply into the valve, and perforate it;
the inflammation spreads to the chordæ tendineæ, and is followe
further ulceration, so as to cause a detachment of the valve segr
the valve is thus rendered incompetent, and with every cardiac co

tion the loose segment, flapping against a part of the auricle, sets up fresh inflammation there, and gives rise to the formation of warty growths on its walls. The loss of substance caused by ulceration extending to parts of the endocardium (chordæ tendineæ, septum) may lead to an aneurysmal bulging of the wall, or even to rupture of the septum or of the heart.

If pyogenetic organisms be the immediate cause of the ulcerative endocarditis small abscesses may form in the tissues of the valves; occasionally one or more larger abscesses are found in other parts of the heart, extending deep into the myocardium; and these again may lead to an aneurysm of the heart, or to rupture into the pericardium. In rare cases the pus may be reabsorbed, and leave scars or calcareous residues (1).

In the more chronic, but sometimes also in the acute cases, we may find deposits of lime salts on the vegetations; but, as a rule, we find these calcareous incrustations in cases in which ulcerative endocarditis has attacked a person already suffering from valvular disease. It is not always easy to distinguish these ulcerations from the atheromatous ulcers due to simple necrosis in a valve with calcareous deposits, the result of either chronic endocarditis or atheroma; in most cases a bacteriological examination will help us to distinguish the two, but not always.

The frequency with which the various valves are affected is shown by the following analysis from the post-mortem records of the Manchester Royal Infirmary (2) for 1891-1895; 20 cases are noted: 7 men and 13 women; average age, 34½; youngest 15, oldest 57. In 15 cases out of 20 previous cardiac disease was noticed: the right side was involved in 1 case only; herein there were vegetations on the tricuspid as well as on the mitral valve: in 7 the mitral alone was affected: in 8 the aortic valve alone. The spleen was found enlarged in 17 cases.

From 1895 to 1897 25 cases were noted: 14 men, 7 women, 3 boys, and one girl; average age, 32; oldest 72, youngest 9. In 20 out of the 25 previous cardiac disease was noticed: in one the right side (pulmonary artery) was affected alone; in 3 both right and left sides; in 6 the mitral valves only; in 3 the aortic only; in 6 both mitral and aortic valves were involved. Splenic enlargement was found in 19 cases (3).

Kanthack and Tickell analysed 84 cases occurring between 1890 and 1897; of these, 51 were males, 33 females; and in all but 16 cases old cardiac lesions were found.

The changes noted in other organs vary considerably, and may be grouped as follows:—

(i.) Primary; such as croupous pneumonia, pleurisy, empyema, meningitis, primary septic foci in the uterus or its adnexa, gonorrhœa, a primary abscess in pyæmia, osteomyelitis, disease of the middle ear, tonsillitis, gastric ulcer, appendicitis, gallstone, etc. (see vol. i. of this work, p. 631).

It must be noted, however, that septic pneumonia may be secondary t infective endocarditis.

(ii.) Lesions due to embolism. These vary as the embolus act simply mechanically or has infective properties: in the first case w meet with infarcts chiefly in the spleen and the kidney, and in th brain; in the brain the area of the blocked artery softens, in per pheral arteries the embolus may lead to gangrene. In the secon case we meet with metastatic abscesses, which may occur either in sma or in very large numbers; and are found in the liver, the spleen, th kidney, the lungs (especially if there be right-sided endocarditis). In th intestines, or even in the stomach, hæmorrhagic infarcts are found, som times of a septic nature infested with numerous micro-organisms, and occa sionally ulcerations of the mucous surface; at other times correspondin to simple infarcts, the intestines present intense congestion, hæmorrhag and even gangrene.

Small capillary emboli are no doubt the cause of the hæmorrhage noted in the skin and subcutaneous tissue, the serous surfaces, the retina and other parts.

3. Lesions which are common to most infectious fevers, and whic may be due to micro-organisms, their toxins, or to the accompanyin pyrexia. Enlargement of the spleen, so-called "cloudy degeneration" o the liver and kidney, and nephritis (in which the kidney is large, pale and shows small hæmorrhages), are included in this group.

Symptoms.—The symptoms of infective endocarditis vary conside ably in individual cases: the heart symptoms may be quite insigni ficant or even absent; as, for example, when acute infective endocarditi complicates a septic disease, as pneumonia, empyema, or meningitis, i which often only the autopsy reveals the endocardial lesions. In othe cases the heart symptoms are more pronounced: this is more particularl the case in the subacute or even chronic form which complicates rheumati endocarditis.

Owing to the great diversity of the symptoms certain types of infec tive endocarditis have been formulated. We may distinguish in the firs place between an acute form and a subacute or chronic form.

The acute form includes the septic type, the typhoid type, and th cerebral type; the chronic form is noticed in old valvular affections o the heart; by some it is called the cardiac type, or, owing to the peculia fever curve which is noticed, it has been named the intermittent febrile o malarial type. We will briefly consider the principal features of thes various types, and then note the symptoms in detail.

(*a*) The *septic or pyæmic type*, which is noticed in puerperal cases an in other forms of septicæmia and pyæmia, includes all the symptoms o a severe septic infection. The onset is acute; with or without preced ing general malaise the disease is ushered in by more or less sever rigors, followed by heat and sweating, which may be repeated after shorter or longer interval; between the rigors the temperature generall remains high, it may, however, be remittent; the skin may show patche of erythema, hæmorrhage, or superficial collections of pus; the pulse is quick and feeble; the respiration is hurried and superficial; nervous symptoms, such as headache, delirium, somnolence, are usually present; at times symptoms of cerebral embolism may appear; the tongue is usually furred, and may become dry and brown; there may be great thirst, anorexia, and vomiting; there is often a good deal of tympanites and diarrhœa. Metastatic abscesses may form in various organs and tissues, but often do not give rise to definite symptoms, as, for example, in the lungs.

The examination of the heart may reveal either no abnormal signs, or audible murmurs; from their presence alone we may not conclude that we have to do with infective endocarditis, for such murmurs are not uncommon in simple cases of pyæmia and septicæmia, without any ulceration of the valves of the heart. Of other symptoms common in ordinary pyæmia I may mention albuminuria, jaundice, and pain and swelling of the joints with suppuration. Death generally takes place within one or two weeks.

(*b*) In the *typhoid type* infective endocarditis resembles enteric fever as regards the general aspect of the patient, the condition of the tongue, which is brown, dry, and furred, the presence of diarrhœa and cerebral symptoms; but we not infrequently see rigors, petechiæ, and optic neuritis—symptoms which are very rare in enteric fever: the heart symptoms in this form again may be absent, or indefinite. The temperature is generally very irregular; rigors may occur throughout the whole duration of the disease, followed by profuse sweating; and attacks of embolism in the brain, kidney, and spleen are not uncommon. The duration of the disease, when assuming this form, varies from two to three weeks; sometimes it lasts longer.

(*c*) *Cerebral type.*—This type is chiefly abstracted from cases of malignant endocarditis complicated with meningitis, either cerebral or cerebro-spinal. The affection begins in these cases with cerebral symptoms—headache, somnolence going on to unconsciousness and coma, or delirium and convulsions. The heart symptoms are less pronounced and often absent. Rigors are not often present, but attacks of embolism may occur and direct attention to the heart.

(*d*) *Cardiac or Malarial type.*—This represents by far the largest number of cases; it occurs in persons in whom the heart has already been damaged by previous disease. It runs, as a rule, a subacute and chronic course, and may last six months, or even more than a year. Though recovery is extremely rare, this variety is not always fatal.

The onset of the disease is generally insidious; the patient complains of general malaise, and has an anæmic appearance. Sometimes an increase of body temperature, with but few other symptoms, may be the first sign of it, as in a case under the care of my colleague Dr. Steell: a young man suffering from an old valvular affection of the heart, whilst in the hospital suddenly showed a rise in temperature, and after a few days manifested characteristic signs (rigors and so forth) of infective endo-

carditis. At other times the affection resembles rheumatic a
pains in the joints and slight pyrexia being prominent features.
these symptoms have lasted a few days, rigors appear, followed l
and sweating. During rigor the temperature may reach 104°
more, and a few hours later the temperature may come do
normal. The rigors occur at irregular intervals: two or thre
occur in one day; at other times several days or weeks may elapse
a second rigor is observed. In a good many cases the rigor is r
by a mere sense of chilliness followed by sweatings; in others, a
remittent or intermittent pyrexia, going on for weeks or
without any rigors, is a prominent feature. Thus, in one case,
saw with Dr. Renaud—a girl, aged 20, who at the age of 16 had
attack of rheumatic fever from which she recovered, but which l
affected with mitral disease—the only noticeable feature was an inter
pyrexia—the morning temperature being 98°, the evening temp
99° to 100°; beyond this no other symptom was noticed, a
patient felt no further inconvenience. This state persisted for o
months, when she had an attack of cerebral embolism. From t
had partially recovered when a second and fatal attack of em
supervened. At the necropsy new deposits were found upon an ol
tion of the mitral valve, and the vegetations showed the prese
numerous streptococci.

A remarkable feature in the cardiac type of infective endocar
the occurrence of embolism. This occasionally affects peripheral a
(posterior tibial, brachial, popliteal, and even abdominal-aorta), bu
often the left middle cerebral artery, or one of its branches, especia
Sylvian artery. The blocking of the cerebral vessels may only p
temporary paralysis or aphasia; but often these attacks are fo
by others which leave a permanent lesion, and most frequently l
complete hemiplegia.

Some of the viscera also may be the seat of emboli; thus
infarcts are not uncommon, which may give rise to no symptom
occasionally certain symptoms enable us to diagnose the infa
namely, sudden pain in the region of the spleen, with enlargement
organ, and occasionally a friction sound over the spleen. It mu
be forgotten, however, that, without the presence of an infarct, the
is often considerably enlarged in infective endocarditis.

Quite as common are *renal infarcts*, which only give rise to sym
when the infarct is large; in such a case sudden pain is felt in the
of the kidney, and hæmaturia and remittent pyrexia appear.

Infarcts of the lungs can be inferred if the patient have a sudde
in the chest, with dyspnœa, followed by the expectoration of sa
len sputum. On physical examination, if the infarct be lar
notice over a small area dulness on percussion, increased vocal fr
bronchial or tubular breathing, and fine crepitations; the temperatu
rises and assumes a remittent character. Pulmonary infarcts freq
lead to embolic pneumonia, and often also set up localised pleurisy.

endocarditis be situated on the right side of the heart we occasionally meet with multiple metastatic abscesses in the lungs, which give rise to no definite symptoms.

Embolism of the mesenteric artery—a rare occurrence—may give rise to severe abdominal pain, with hæmorrhage from the bowels and grave general disturbance leading to collapse.

Other symptoms often noticed are pronounced anæmia, which may be present from the beginning: examination of blood shows the red blood corpuscles to be diminished; the leucocytes are often increased, and a few eosinophile cells may be detected. Bacteriological examination of the blood reveals the presence of micro-organisms, notably streptococci.

Petechiæ and hæmorrhage from the mucous membranes are occasionally noted, the latter more particularly when the aortic valves are affected.

Pains in the joints are often complained of; in many cases the joint is neither swollen nor reddened, and the affection is probably of a toxic nature; at other times we meet with a definite arthritis, or again, with suppuration of the joint.

Hæmorrhages in the retina and *optic neuritis*, according to some observers, are of common occurrence. I have seen hæmorrhage more frequently than optic neuritis.

Enlargement of the spleen is very often noticed, and may reach a considerable degree, so that the spleen can readily be felt; it is not a constant symptom, however, and in some cases the spleen, as shown by the necropsy, is even smaller than in the normal state.

The *liver* is sometimes found enlarged, and jaundice may be present. In rare cases the liver appears diminished, and the case may simulate acute yellow atrophy. The occurrence of infective endocarditis in persons suffering from gall-stones has already been alluded to when speaking of the pathology of the disease (vol. i. p. 631); this may occur in persons who have not had rheumatic endocarditis.

The *urine* often shows traces of albumin and blood, and the presence of casts, both epithelial and granular.

The *bowels* are often constipated; occasionally we meet with profuse diarrhœa, and sometimes (see above) with hæmorrhage from the bowel.

The ordinary *complications* are pneumonia, pleurisy, pericarditis, aneurysm, cerebral hæmorrhage; this last was noticed in two cases which occurred in the Manchester Infirmary; an embolus was carefully searched for, but with negative results.

The symptoms which relate to the heart are well pronounced in the cardiac form, and we meet with the signs of mitral or aortic disease, or of both; in rare cases we have evidence of an affection of the valves of the right side of the heart. There is nothing in the character of the bruits or in the size of the heart to enable us to diagnose infective rather than benign endocarditis; during the course of the disease the murmur may undergo some change, but this may also occur in rheumatic endocarditis. The presence of right-sided valvulitis is of greater diagnostic value, as it is of very rare occurrence in the rheumatic or benign endocarditis. authors lay stress on the loudness of the murmurs, on their (metallic) character, and on the propagation of the mitral murmu axilla and angle of scapula; but these signs are also noticed in th form of endocarditis. Subjective symptoms, such as palpitati over the region of the heart, excessive dyspnœa, have no di value.

As already stated, the cardiac form of infective endocarditi always runs a chronic course; occasionally it may occur in a form. When treating of the pathology, I mentioned one inst which, previous to the occurrence of infective endocarditis, there had been a ruptured aortic valve. Recently I saw, with Mr. of Blackley, a case of infective endocarditis in a compositor, a who had always enjoyed good health, and who had never been with rheumatism; he was suddenly seized with a rigor whil work; he was brought home, and his wife, who had been took his temperature and found it 103·5°; in the course of a fe the temperature was again normal, and the patient felt quite wel morning after, he had another rigor and rise of temperatur in the evening he had still another rigor. When I examined the soon after, I found the temperature normal, and the patient com only of some oppression; the heart's action was somewhat tum and the arteries beating rather forcibly; over the aorta a faint bruit was audible. The spleen was enlarged. The patient h taking quinine, and now some arsenic was added to this; the however, continued for two days, when the patient suddenly looked upon this case as one of idiopathic acute infective endocar

The above types by no means represent all the clinical f infective endocarditis. Thus it is found in association with pne in which case there is very often no special symptom to lead suspect its presence. It may occur with gonorrhœa, in which c heart symptoms are often pronounced, whilst septic symptoms obvious; and, lastly, we meet with cases in which the distinction rheumatic and infective endocarditis is impossible.

Diagnosis.—In spite of our improved clinical methods, and t cation of bacteriology to clinical medicine, the diagnosis of infec docarditis is still often a matter of difficulty.

Enteric fever may be distinguished from infective endocar the mode of onset, the temperature curve, the roseolar spots, tym and so forth. [See "Enteric Fever," vol. i. p. 836.] Repeated ri rare in enteric fever, and cardiac murmurs seldom appear at th ning of it. In doubtful cases Widal's serum test may be a usef if, after the sixth day of illness, this test give negative results fever may with great probability be excluded; on the other h peated rigors, and especially the occurrence of attacks of embolis most strongly for infective endocarditis.

From septic and pyæmic infection, unless heart symptoms nounced and signs of embolism are present, the disease is not easily distinguished. This will be easily understood, for infective endocarditis is indeed nothing more or less than a septic disease with the special localisation of the micro-organism in the heart valves. Bacteriological examination of the blood (see below) commonly shows us the presence of septic micro-organisms; and the same observation applies to the meningeal or cerebral form. It is only in cases of tuberculous cerebro-spinal meningitis that the withdrawal of fluid by means of puncture of the spinal membrane in the lumbar region—which would show the presence of tubercle bacilli—can be of any diagnostic value; as the same organism that is found in non-tuberculous meningitis, be it suppurative or cerebro-spinal, has been found in infective endocarditis.

In the cardiac form, when the heart symptoms are well pronounced, several signs help us to distinguish between the rheumatic (or benign) and the malignant endocarditis. These are :—

(i.) The presence of pyrexia.—This is often one of the first symptoms, and may show the remittent or intermittent type; should the pyrexia be accompanied by rigors occurring at irregular intervals and not affected by either quinine or arsenic, the diagnosis may be looked upon as almost certain.

(ii.) The anæmic appearance of the patient.—Anæmia often follows the first attack of rheumatic endocarditis; but the persistence of anæmia for a long time, or the occurrence of anæmia long after the attack, should certainly make us suspect malignant endocarditis.

(iii.) Enlargement of the spleen has already been discussed on p. 881.

(iv.) Changes in the retina, whether in the form of optic neuritis or of small hæmorrhages, when occurring in persons suffering from endocarditis, are indicative of the infective form; and it is well to examine the eye in all cases of endocarditis.

(v.) Hæmorrhages in the skin and from the mucous membranes.—Epistaxis is a common symptom in rheumatic endocarditis when the aortic valves are affected; and hæmoptysis is frequently noticed early in mitral disease, and at a later stage in other heart affections. Hæmorrhages into the skin and subcutaneous tissue, on the other hand, due probably to numerous small capillary emboli, are indicative of infective endocarditis. Of hæmaturia from renal infarcts and of melæna from infarcts of the mesenteric arteries I have already spoken; but in themselves, and without other signs of infective endocarditis, these hæmorrhages are of no diagnostic value, as they may be the result of the chronic venous congestion secondary to chronic endocarditis.

(vi.) Bacteriological examination of the blood.—Many are the observations on this subject, and various the methods which have been devised to obtain sufficient blood for the culture of micro-organisms. Petruschky uses the blood obtained by cupping. Lithmann withdraws about 5 c.c. of blood directly from a vein of the arm by means of a sterilised syringe. A portion of this is mixed with agar-agar which has been previously liquefied in a water-bath at a temperature of 40° C., and the mixture is poured out into Petri's capsules to secure cultivati the micro-organisms present. In the acute septic cases numerous cu of streptococci and other cocci are found; in the chronic cases, thoug case may be one of infective endocarditis, this method does not a show the presence of micro-organisms. Of three chronic cases of inf endocarditis H. Cohn found a few colonies of staphylococci in one In several cases of chronic infective endocarditis under my own ca which the diagnosis was verified by the autopsy, some venous bloo aspirated after the method of Lithmann, and examined bacteriolog by my colleague Dr. Delépine, but with negative results.

Prognosis.—The prognosis of this disease is in all cases very The acute form, be it of the pyæmic, typhoid, or meningeal type, is a invariably fatal, death taking place sometimes within a few days. E gives the case of a man who began with typhoid symptoms, soon foll by coma and hyperpyrexia; the case ended fatally the next day. aortic valves showed ulcerations, and a metastatic abscess was four the brain. In other cases the symptoms may go on for several The chronic or cardiac form may last for months and occasionally year; yet a fatal termination either by exhaustion, embolism, or com tions is the rule: several recoveries of undoubted cases have, however, recorded. When speaking of the pathogeny (vol. i. p. 632), I ment a case in which malignant endocarditis occurred after an injury, a which the patient recovered with a damaged aortic valve, and is a present time in a satisfactory state of health. Another patient, wh saw with Dr. Hassall of Northwich, with all the signs of infective carditis implanted on a diseased aortic valve, recovered.

Treatment.—Many are the drugs that have been recommend infective endocarditis. Apart from the general treatment with t stimulants, and rest, the same drugs as are given in rheumatic carditis—such as the alkalies and salicylates, antipyrin, phenacetin so forth—have been recommended, but the results have not bee couraging. Large doses of quinine appear more useful, though quinine does not prevent the occurrence of the rigors, even in doses. Fraentzel recommends large doses of quinine with arsenic I have for some years given this combination; yet, except in the cases quoted above, and in a third case in which the sympton endocarditis occurred after an attack of gonorrhœa, and in there was also a peri-urethral abscess, the fatal termination wa averted.

Benzoate of sodium, recommended by Kleber and others, ha given any good results in my hands.

Sulpho-carbolate of soda (half-drachm doses) is recommended b Sansom, who records one case in which, when death took place later period, distinct cicatricial tissue was found at the site of th ulcerations.

The subcutaneous administration of antistreptococcus serum has recently recommended; judging from the successful cases publishe

Sainsbury and by Pearse, this treatment deserves a trial. Sir Douglas Powell has tried in five cases subcutaneous injection of yeast, but without any marked result; and in one case nuclein was used, which caused a temporary fall of the temperature.

J. Dreschfeld.

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For further references the reader is referred to the article on this disease in vol. i.

J. D.

DISEASES OF THE MYOCARDIUM

As with other muscular organs, the heart is liable to fatigue, to overstrain, to disturbed innervation, to impaired nutrition; either, in the first place, from defect in the nutritive qualities of the blood with which it is supplied, or, in the second place, from temporary or permanent restriction in that supply through temporary or permanent alteration of the vessels. Further, the heart muscle may undergo degenerative changes, or may atrophy and be replaced by fibrous tissue; and this degeneration or atrophy and fibrous replacement may be general or localised. Yet, again, the heart muscle may undergo physiological hypertrophy in obedience to the demands of excessive labour, and this condition, although not one of disease, has to be reckoned with, since it leads to textural changes; finally, the heart may be invaded or occupied by growths, parasitic or other, of various kinds. With the various diseases of the endocardium, pericardium, and valves of the heart I have here no immediate concern, although I shall have to refer to them incidentally in an endeavour to give a clear account of myocardial lesions.

The several lesions of the myocardium above mentioned will be found to group themselves naturally under the pathological headings of—

I. Impairment secondary to general blood conditions.—(A) Anæmia; (B) Toxic changes.

II. Impairment secondary to altered blood-supply.—(A) From paroxysmal affections of coronary arteries; (B) from permanent changes in coronary arteries; (i.) Atheroma; (*a*) fatty degeneration; (*b*) fatty infiltration; (*c*) fibroid infiltration; (*d*) aneurysm of the heart; (ii.) Thrombosis or embolism; (iii.) Aneurysm.

III. Impairment due to senile changes.—(*a*) Pigmentary deg tion; (*b*) atrophy.

IV. Impairment arising from functional strain.—(*a*) Hypertr (*b*) acute dilatation; (*c*) textural damage.

V. Impairment of inflammatory origin—Myocarditis.—(*a*) stitial; (*b*) parenchymatous; (*c*) purulent; (*d*) syphilitic.

VI. Growths.—(*a*) Sarcoma; (*b*) myxoma; (*c*) fibroma; (*d*) gu (*e*) carcinoma; (*f*) lipoma; (*g*) cyst; (*h*) myoma; (*i*) tubercle.

VII. Parasites.—(*a*) Hydatid; (*b*) cysticercus cellulosæ; (*c*) a mycosis; (*d*) trichina spiralis.

I. IMPAIRMENT SECONDARY TO GENERAL BLOOD CONDITIONS

Anæmia.—*Pathology.*—In cases of marked anæmia, as in chlorosi nutrition of the heart muscle suffers; the organ is paler than na somewhat glistening and wet-looking on section, and gives less tha normal resistance to the pressure of the finger. On micros examination in persons who have died from some intercurrent m no change may be noticed; but most commonly the fibres have u gone a certain degree of fatty change, and present a few refra granules. In some cases of extreme anæmia, however, a very no degree of fatty change may be found in the muscular fibres; the int surface of the organ, especially over the left ventricle and pap muscles, presents a streaked or flecked appearance, due to groups of opacities seen through the transparent intima, the degeneration affe the muscular fibres having a patchy distribution.

Although the fatty heart is always somewhat increased in size, it not be increased in weight; the specific gravity of muscles being re by fatty change. The pericardium and endocardium usually e change, but the cavities of the heart are enlarged, especially the ventricle; and slight incompetence of the mitral valve is often rev when the valve is properly tested by a fluid pressure equal to that o blood. I have often seen a heart inadequately tested in this respect degree of regurgitation, clinically observable, may be overlooked i ventricle and valve are not subjected to sufficient fluid pressure.

On microscopic examination, groups of fibres are found in whic fibrillæ are more or less replaced by rows of refracting fatty granule change appearing first in the neighbourhood of the nuclei of the f Besides the groups of more intensely fattily changed fibres, the fibres are more or less dotted with fatty granules.

Clinically, in all cases of extreme simple anæmia of any conside duration, one may observe a certain degree of enlargement of the h the apex beat is a little to the left of the normal, and the area of percu dulness extends slightly upwards; frequently a soft murmur is heard over the apex beat, which is not merely conducted from pulmonary area, but has the characteristics of mitral regurgitation, a no doubt due to a dilatation of the left ventricle, so that the ba attachment of the papillary muscles becomes displaced, and the valve slightly incompetent at the moment of greatest intra-ventricular pressure. The heart's action is quickened, and is peculiarly irritable to the calls of slight effort or to reflex or emotional stimuli. These symptoms, which constitute the cardiac features of anæmia, are of course only in part directly due to the state of the heart muscle, they depend rather upon the condition of the blood and the debilitated state of the nervous system; and to both these latter causes, as well as to the cardiac enfeeblement, is also attributable that degree of œdema of the extremities which is so common in marked anæmia.

B. Toxæmia.—*Hyaline degeneration.*—A peculiar hyaline swelling of the muscle fibres of the heart in diphtheria has been described by Bouchut, Labadie, Lagrave, and Rosenbach. The last-named author looks upon it as an inflammation. Similar changes are met with in the voluntary muscles in typhoid fever. Boyce speaks of it as a hyaline degeneration of connective tissue, consisting of hyaline material similar to amyloid, but without the chemical reaction of the latter. Hyaline degeneration identical with that in the myocardium is more commonly observed around the arteries, sometimes permeating, and causing extensive atrophy of the muscle fibres of their middle coat.

Cloudy swelling is a condition in which the fibres of the heart lose their striation and become finely granular; it is met with especially in diphtheria, typhoid and typhus, and is indeed common to all febrile states of sufficient duration.

Fatty degeneration.—In certain poisoned conditions of blood, as from lead, arsenic, and, in a most notable degree, from phosphorus, fatty degeneration of the muscular fibres of the heart may be very extensive; and, in cases of phosphorus poisoning in which the patient has survived the more immediate gastro-intestinal symptoms, it is the principal source of danger. The mildest form of blood contamination—although very important from its being so common—is the absorption of ptomaines from the colon in neglected torpidity of the bowels, a source no doubt operative in the production of the fatty heart of anæmia. The most intense of the poisons of organic origin affecting the heart is that modification of the toxine of diphtheria which is formed in the later stages of this disease, and which appears to be responsible for that profound fatty degeneration of the heart (in common with other organs) which is only equalled in cases produced by phosphorus.

Repair in fatty degeneration.—Clinical observations would lead us to suppose that repair of fattily degenerated hearts is possible, and even of frequent occurrence; Dr. Coats is of opinion that it takes place by absorption of the fat and an actual new formation of muscular tissue. That such new formation is abundantly possible is evident from the readiness with which healthy hypertrophy is established to compensate valvular defects, or in response to other unusual calls upon the muscular activity of the heart.

Under the heading of changes of the myocardium of toxic origin we should certainly include those consequent upon chronic gouty conditions and chronic uræmic poisoning; although, as in the less defined c induced by alcoholism, nicotinism, and the like, the lesions have feat common with those induced by other causes, and will be described

There can be little doubt that the high-pressure pulse and cons increased call upon the heart which are associated with chronic aff of the kidney are combined effects of central nervous induction, for their purpose such an increase of blood-pressure as shall p compensatory kidney function. In chronic gouty conditions the vascular function is similarly modified; and in other cases of h high arterial blood-pressure from mental strain or other causes th effects, although less in degree, are observed in the myocardium. effects are, first, hypertrophy; and, secondly, fibro-fatty degenerati

II. IMPAIRMENT OF THE MYOCARDIUM SECONDARY TO A BLOOD-SUPPLY.—A. **Paroxysmal conditions of coronary arte** Many authors have pointed out the occasional occurrence of angina p in young people attributable to excess in tobacco-smoking; an observed the anginal paroxysm of like causation in older p Besides its other effects tending to disturbance of the cardiac inner Dr. Huchard holds the view that nicotine has a more direct acti causing spasmodic contraction of the smaller vessels, and, in these especially of the coronary vessels. It is difficult to bring ev sufficiently demonstrative to prove this opinion or to refute it Huchard relies chiefly upon the spasm of voluntary muscle upon the pallor and arterial contraction observed in nicotine in tion, upon the high arterial tension often to be observed in sm and upon the experiments of Claude Bernard in 1857, and b self and others since, showing the local effect of nicotine in c contraction of the vessels in the frog's foot. There is every rea believe that the coronary arteries, like other vessels of equal si equally richly endowed with muscular tissue, are liable to spa contraction; and it is quite possible, as maintained by Huchard, t some cases the abuse of nicotine may directly cause such constricti produce temporary anæmia and disturbed function of the heart r It has not been shown, however, that any textural damage to the substance has been caused by the vaso-motor effects of nicotine u circulation. Of course, the remoter effects of nicotine in causing a and muscular degeneration, if such there be, are not included present subject.

B. Permanent changes in the coronary arteries.—(i.) **Ather the coronaries.**—This may arise: (*a*) From the natural effects leading to degeneration of the intima, with secondary thickenin softening, or calcareous deposition.

(*b*) These senile changes may be anticipated by constitution ditions, especially syphilis, alcoholism, and gout; the sequence of being much the same, namely, degenerative impairment of ela patchy thickening, fatty change, or calcareous deposition.

(*c*) Hereditary disposition plays an important part in determining premature decay of the arterial system.

The above conditions are general to the whole arterial system, but are most manifest at those portions of it at which the stress of normal arterial pressure is most heavy. The origin and arch of the aorta and the coronary arteries are the portions thus affected which concern us at the present moment; and it may be noted that atheromatous narrowing of the coronaries is generally most marked at their aortic origins, and is often limited to these parts.

(*d*) The chronic arterial strain of laborious occupations has a very important influence in producing chronic patchy endarteritis of the aorta and the coronary arteries; and it operates very commonly in conjunction with the causes of arterial degeneration spoken of under headings *b* and *c*.

There can be little doubt that the peculiar patchy distribution of endarteritic thickening is due to small rifts at points of least resistance of an intima rendered more brittle by degenerative changes, and to the secondary nuclear overgrowth and subsequent degenerative changes ensuing thereupon.

(*e*) Apart from the general effect of syphilis in disposing to arterial atheroma, syphilitic granulomata may form in and about the arteries, especially thickening their inner coats, and thus often leading to narrowing or obliteration. [*Vide* art. "Disease of Arteries," vol. vi.]

(*f*) Vessels of small calibre, such as the coronary arteries when narrowed, and with their intima changed by atheromatous or specific arteritis, are very apt to become abruptly and completely closed by thrombosis.

(*g*) The coronary arteries, like other vessels, are liable to embolic closure, although they are much less prone to this obstruction than are other vessels more directly in the current of the circulation. Such embolisms when they arise may be simple or septic.

Having now enumerated the possible causes of narrowing or obliteration of the coronary arteries, let us look to the consequences of such narrowing, which we shall find to embrace the most important lesions of the cardiac muscle.

(*a*) *Fatty degeneration of the heart.*—A gradually increasing impairment in the blood-supply of the heart, and a correspondingly diminished thoroughness of the irrigation of its tissues with blood, are the most frequent causes of fatty degeneration of the muscular fibres. I have already spoken of fatty degeneration of the heart as a consequence of general anæmia, and in certain states of toxæmia; the degeneration arising from local anæmia, due to constriction of the supplying vessels, is of the same kind, but is much less acute, and is more patchy in its distribution. In hearts in which the coronary narrowing affects both vessels at their origins, the distribution of fatty change would be more uniform; but these cases are rare. Often only one coronary vessel is thus affected, and sometimes only certain branches within the substance of the heart are much contracted by atheroma. Thus the change, at least in any serious degree, may be limited to one side of the heart, or or more portions of one or both ventricles or auricles.

The change, for reasons to be mentioned immediately, besides much more insidious and slow in its progress, is mingled with changes and, in particular, with false or fibroid hypertrophy of the In very old people, in whom the whole process is one of senile the fatty degeneration may be simple and unattended with changes.

The process of fatty degeneration of the cardiac muscle consi already stated, in the gradual replacement of the sarcous elemei fatty granules, the deposition of granules beginning about the nucl extending linearly towards the fibre-ends. The affected tissue i rendered more opaque in streaks of a tawny yellow colour, is soft more friable under the finger, and in well-marked patches gives a section. In some cases, in which the degeneration is extreme restricted area corresponding with an occluded vessel, the fatty sof may be so great as to resemble abscess. It is said (11*a*) that in cases the fatty change proper is preceded by a "cloudy swelling," in the fibres become finely granular from the deposition in them protein granules, which are to be distinguished from fatty granul only by their more dim and cloudy outline, but also by their react strong acetic acid or caustic soda or potash, either of which oblit them, whilst the same reagents bring out the granules of true degeneration in stronger relief. Both the protein and fat gr are derived from the sarcous elements of the muscle which they r As the disease advances the striation of the fibres becomes graduall at first at the extremities of the fibres, finally towards their n centres.

In combination with the fatty degeneration there is more atrophy of the muscular fibres, and in substitution for them an growth of connective tissue elements resulting in the formation of tissue (fibroid or false hypertrophy). In this respect fatty degene of the heart, induced by restricted blood-supply from narrowed v differs from the same degeneration due to general anæmia or toxic I have already indicated an exception in the case of old people, in the degenerative changes are a part of general senile decay.

Although the internal surface of the ventricles may be specke streaked with opacities—much more irregularly disposed, however in the case with degenerations of general blood origin—the endoca itself is rarely affected. The size and weight of the heart, and the ness or thinness of its walls, depend chiefly upon the amount of substitution which is associated with the fatty change. The perica is not necessarily involved, although it may be more opaque and t than normal.

It was found, in speaking of the more acute fatty degene of the heart due to general blood conditions, that partial or cor repair was possible by a renewal of the muscular fibres in the sam as an extra growth of such fibres can take place in healthy hypertrophy, whilst at the same time the fattily degenerated fibres became absorbed. In degeneration due to permanently narrowed blood-supply, however, no such repair can take place to any appreciable extent; for the anastomosis of the two coronary arteries, supposing only one to be affected, is not free enough to provide a sufficient circulation for the purpose. Nevertheless, we may see in the overgrowth of fibrous tissue, of a somewhat depraved sort it is true, an attempt to maintain the due resistance of the heart walls to blood-pressure, without however any corresponding preservation of contractile power.

Symptoms and signs.—The fatty heart is a weak heart, weak in its muscular power, and weak in its resistance to blood-pressure. It is either more or less arrhythmic in action, or readily becomes so under any extra demand upon it from excitement or effort. It is also (except in cases in which the degeneration of the heart goes hand in hand with general atrophy of blood and tissues in old age) an enlarged heart, increased in size by the dilatation of the ventricles, and especially of the left ventricle, under the normal blood-pressure; and often increased in size also by false (fibroid) hypertrophy. Hence, in a person usually beyond middle life, with a feeble circulation and a tendency to blueness of the extremities, if we find the superficial dimensions of the heart increased, the apex more to the left than natural, the dulness extending an interspace higher, and perhaps a finger's-breadth more to the right than is proper, and if on auscultation we find a marked indistinctness of the first sound and an irregularity of beat both as regards time and force, we may be sure of degeneration of the heart, and that the degeneration is more or less fatty.

A very common symptom is the occasional occurrence of attacks of syncopal or anginal failure [see "Angina Pectoris," vol. vi.], but as these attacks are often simulated by those of a much less serious nature, they are always to be taken into account in conjunction with the signs of a cardiac enlargement. In cases of anginal attack attendant upon organic change in the cardiac muscle, and especially when such change is associated with coronary stenosis, the immediate cause of the earlier attacks is generally some increased call upon the heart from excessive exertion, such as walking quickly or uphill; and the result is to bring the patient to a stop at once. Later attacks may occur when the patient is at rest or asleep. In anginal seizures, when the heart is sound, the patient often tends to move about, and if the attack be not very severe it may not prevent the continuance of walking or other exercise.

In advanced cases of fatty heart, cases in which more distinct anginal symptoms may not have occurred, an altered respiratory rhythm is not infrequently to be observed, which is especially apt to occur during sleep; namely, an increasing shallowness of breathing down to absolute cessation for 20, 30, 40 seconds, then several profound and heaving respirations take place which again gradually subside to complete pause (Cheyne-Stokes breathing). During the pause the patient generally wakes up with a start, and his sleep is thus much interfered with and becomes reduced to a succession of short dozes. The peculiar br is to be observed during the waking hours also. The pulse co with its usually irregular action practically unaltered duri arrhythmic breathing and pause ; it is to be noted, however, that cases during ordinary or deep breathing the pulse is distinctly during the inspiratory wave. It must, lastly, be confessed that ra are met with in which, even with a marked degree of fatty heart, are discovered up to the moment of fatal syncope or angina. I mu my belief, however, that if the opportunity presents itself for a examination of such cases, and the possible presence of emphys taken into account as masking an increase of the cardiac area, the evidence of fatty degeneration is rarely to be missed.

The disease is most common at or beyond 50 years of age. M frequently suffer than women in the proportion of nearly two to one (All the functions of a person with fatty heart are performed in a manner. He is the subject of atonic dyspepsia, with a great tend flatulent distension of the stomach ; his bowel and liver functi torpid ; the urinary secretion, very sensitive to external surface t ture, is of low range of specific gravity, and often contains a t albumin. The brain is easily fatigued, the temper irritable. gentle level exercise can be taken with comfort.

Treatment.—The treatment of fatty degeneration of the heart altered blood-supply is a matter of great importance, hence the n of recognising the lesions at the earliest possible stage.

In the earlier stages regular exercise short of fatigue, and ada a person in whom a weakness of the central organ of the circul recognised, is of importance ; quiet walking on the level, ridi hunting), cycling (avoiding hills), driving, sailing, quiet rowing, be allowed ; and gentle incline walking, adapted to the case, taken as prescribed exercise. Covert shooting may be followed, rough walking or hill shooting. Golf and croquet are games well to such people. For these early cases, a course of Nauheim bat exercises may be taken with advantage from time to time, the e being especially valuable in aiding by tonic muscular contractions th of blood to the heart, disgorging the venous circulation, and thus the forward movement. Cold bathing should be forbidden, and a given against walking against cold winds.

A nutritious diet, rather nitrogenous than fatty or starchy, allowed, distributed in three regular meals daily, eaten slowly, and in quantity to the lessened requirements of a less active life. A m amount of wine is usually required.

Arsenic, iron, and strychnia are the tonics especially valuab they should not be given in more than two, or at most three dos for short courses—the most careful regard being given to avoid d disturbances. In all cases of lowered blood-pressure there is a te to passive congestion of the organs and especially of the liver, a mild dinner pill and an occasional mercurial alterative are desir

In advanced cases of fatty degeneration the same general plan must be followed still more carefully, and with narrower restrictions as regards exercise, which should only be allowed on smooth level ground, all stair-climbing being strictly forbidden. The diet must be closely watched, especial care being taken to avoid overloading of stomach and acute dyspepsia, as many fatal seizures are attributable to gastro-intestinal disturbance. Persons with fatty heart are extremely sensitive to external cold, and should be clothed very warmly. A thorough rest, lying down, once or twice a day should be enjoined; the best times to select are before meals; a short rest being taken before luncheon, and a more prolonged rest, of one and a half to two hours, before the late dinner. Such patients should only use warm water for bathing, and for them the Nauheim baths and exercises are not to be recommended.

To a strychnia and arsenic or iron tonic some digitalis, strophanthus, or convallaria may be added; and an aromatic stimulant and carminative draught should always be at hand in case of syncopal attacks, and may often usefully contain a little nitro-glycerine.

Finally, in cases of fatty heart which have advanced to the production of any decided symptoms, the employment of oxygen inhalations twice or three times a day is valuable as a cardiac restorative; it operates principally, no doubt, in stimulating cardiac nutrition and in facilitating the removal of waste tissues from the organ by flushing it with more highly oxygenated blood. For the Cheyne-Stokes respiration, in advanced stages, there is no more powerful means of affording relief than oxygen inhalations in combination with strychnia. In such cases, however, it should not be employed with the naso-oral inhaler, but a current of oxygen should simply be played over the mouth and nostrils of the patient for five or ten minutes without any extra respiratory effort on his part.

(β) *Fatty infiltration of the heart.*—Fatty infiltration of the heart is a condition in which, owing to the deposition of fat in the interstices of the muscular fibres, these fibres themselves are compressed, impeded in action, and become atrophied.

A certain amount of adipose tissue is naturally present on the heart, especially along the superficial course of the coronary vessels and in the sulci at the base; under certain conditions this tissue develops in inordinate quantity and spreads over the cardiac surface, penetrating, chiefly with the arterial branches, into its muscular substance. This increase and extension of the adipose tissue is most marked over the right ventricle, and may constitute a layer of considerable thickness which by its encroachment upon and between the muscular fasciculi may cause their atrophy and replacement, and thus considerably weaken and embarrass the heart. The extension is always from the subpericardial surface and chiefly along the arterial lines. The atrophy of the muscular tissue which attends upon fatty infiltration is for the most part consequent and secondary; but it is probable that in some cases a primary atrophy of the muscle leads to the secondary development of fat in the connective tissue—which is everywhere present and potentially fat-bearing.

Thus, clinically, we have two forms of fatty infiltration of th the one in which the fat is rapidly stored and extends into and en upon a higher tissue, the function of which it embarrasses, and tl tion of which it mechanically interferes with; the other in wl fatty tissue merely, as it were, fills up the interstices left by an ing muscular tissue.

Of these varieties the first is by far the more common and im It is met with in persons of inactive and often indolent and self-ir lives, in men at middle age, in women towards the climacteric p soon after it. It is the people who have good appetites a primary digestion with faulty assimilation and inadequate eli power that are especially liable to this disease. Indulgence in and especially in malt liquors and the sweeter wines, certainly fa occurrence; and there are certain maladies upon which it is p liable to ensue, especially those affections which involve a depriv respiratory surface, such as chronic emphysema, or fibroid disease lung in old-standing quiescent phthisis, or secondary to pleuritic or unresolved pneumonia, etc. Defective elimination by the bow kidneys is similarly efficacious. It must be carefully remember ever, that no organic disease of any kind is necessary as the for of this affection, which may arise solely from an excess of ali supply over demand, however this may be brought about.

Symptoms and signs.—Persons thus affected are stout, increa weight, with a thickening layer of adipose tissue, full abdome often tender livers. Their circulation is feeble and usually quicker than was normal in them. There is some excess of ven their colouring, they are short-breathed on exertion, and sweat Later they manifest functional disturbances of the heart's action, induced on exertion or coming on without it. The cardiac du increased by an interspace upwards, but the apex beat is difficult and the cardiac impulse tends to be more felt towards the epig than beyond the normal position to the left. The sounds are le than natural, otherwise unchanged. There is no change to be fel arteries; the pulse is usually soft, of low pressure, and, if full, pressible. Of course this condition of pulse may be varied b intervening states, such as gout, to which, however, these people peculiarly liable. The urine varies, but is habitually pale and and of rather low than high range of specific gravity.

Whilst in a far less dangerous condition than that attendant truly fattily degenerated heart, these patients are nevertheless ve to succumb to acute disease of any kind, and particularly to br pneumonia, enteric fever, or surgical injury.

The treatment is simple, rational, and, if loyally followed, very su The dietary must be mainly nitrogenous, all superfluous starches, and fats being discarded. Only claret, moselle, or equivalent quan spirit well diluted must be allowed, and in sparing quantity. Th should be at regular times, slowly eaten and strictly moderate in q

But little fluid should be taken with the meal, but tissue change duly ensured and thirst satisfied by a moderate quantity of hot or cold fluid slowly sipped about a quarter to half an hour after the meals, or, sometimes better still, half-way between the meals. A tumbler of hot water with a little fresh lemon juice may be taken at bedtime or in the early morning. Raw fruits, root vegetables, and bread must be avoided, or only very sparingly taken. Daily walking, riding, or cycling exercise must be imperatively enjoined; for the advantage of regulated exercise is not merely to quicken muscular nutritive changes, and so to convert the food taken into proper force-yielding material, but to deepen respiration and to promote the respiratory and other eliminative functions. Hence dumb-bell, fencing, or other home exercises carried on indoors, although they may be useful supplementary aids, are not adequate to replace open-air exercise. Medicinal treatment is of quite minor importance, and may be limited to promoting due elimination, and giving a heart tonic if needed. Turkish baths, or a course at Homburg, Carlsbad, Marienbad, Harrogate, or Nauheim, may be suggested in appropriate cases.

The other form of fatty infiltration attendant upon atrophy of the heart is met with in an altogether different type of individual, one who commonly is already the subject of some grave organic disease, such as tuberculosis or cancer; and its importance and treatment are both merged in the graver malady.

(γ) *Fibroid infiltration of the heart* (Fibrous transformation, Coats; Fibroid degeneration of the myocardium, Orth.; Myocarditis productiva or interstitial myocarditis).—This condition essentially consists in the separation and replacement of the muscular fibres of the heart by an imperfect fibrous tissue generated by overgrowth of the connective tissue of the organ. It is very closely analogous to fatty infiltration, and it cannot rightly be described as a degeneration of the myocardium. It would seem, therefore, that the term "fibroid infiltration" most fitly describes the morbid state present; interstitial myocarditis is also a fairly accurate term, although it conveys a false impression of the disease being an inflammatory one, which it rarely, if ever, is.

Fibroid infiltration of the heart may be described as general and local, although even in general infiltration the disease is not uniformly distributed.

Causes.—Besides the coronary obstruction general fibroid infiltration has another principal cause; namely, chronic congestion of the heart from mechanical impediment to the return of blood from the cardiac veins. This cause is chiefly met with in cases of old-standing emphysema, and in cases in which the whole or a large portion of one lung is the seat of cirrhotic change from old pleurisy, unresolved pneumonia, or fibroid phthisis. Extensive narrowing and destruction of pulmonary vessels and impairment of that inspiratory aid to the cardiac circulation which obtains in healthy respiration, results in a difficulty in the pulmonary circulation, at first overcome by greater diligence of the right heart, but gradually increasing until the venous return to the right auricle is seriously imp A chronic congestion of the walls of the heart ensues, most mark the right side, but involving the left also; and, as a result of this cl congestion, overgrowth of connective tissue and atrophy and degene of the cardiac muscle proper. In the more advanced stages of n stenosis and regurgitation the same conditions are to be observed, h similarly a mechanical origin.

Nevertheless the most important cause of general fibroid infilt of the heart is the obstructive disease of the coronary arteries at o their origin from the aorta, under which head we now consider i more marked degree of fatty degeneration of the muscular fibres i with in association with fibroid infiltration arising from this cause, degree of blood irrigation which will suffice for the overgrowth inferior tissue, such as connective tissue, will not suffice for the nut maintenance of a tissue of such activity, and requiring such fre renewal and restoration as the muscular tissue.

It is thus to be remarked that hearts which are the seat of ge fatty degeneration from coronary obstruction (except quite as a change) are always large hearts, the seat of so-called false hypertr and this it is which furnishes us with an important clue to their cl diagnosis. The increase in size is partly due to increased thickness cardiac walls, in part to dilatation of the cavities of the heart; for fi infiltration, although it increases the toughness of the cardiac diminishes its resilience and contractile power; hence a gradual yi to the blood-pressure, each stage of which is permanent.

Fibroid infiltration as a local affection of the heart arises fron Local obstruction to the circulation, due to local plaques of thick and degenerative constriction of the coronary branches. The h substance may be the seat of innumerable patches of grayish fibroid infiltration from this cause, or there may be one or two patches of larger dimensions corresponding with the territory of a l branch.

2. Corresponding with well-marked patches or "scars" in heart's substance there will often be found a complete occlusion coronary branchlet from thrombosis or embolism, and in an earlier the more distinct signs of a hæmorrhagic infarct may be seen.

3. It is very possible that some of the heart scars which are f may be due to a fibrous repair of partially ruptured fibres.

4. An extensive, although usually superficial fibrous infiltrati the heart may ensue upon pericarditis and adherent pericardium change beginning in the subpericardial tissue and extending more o deeply into the muscular interstices of the heart. Such change started by direct inflammatory irritation, and are often accompanied a certain degree of fatty infiltration.

Pathology.—The minute pathology of fibroid infiltration of the is the same, other things being equal, as that of the same process t place in any other organ; that is, it begins with a proliferation o nuclei of the connective tissue, so that in the earliest stage, rarely observed except at the margins of extension, areas or groups of crowded nuclei are to be seen which are gradually transformed into fibres; these again in their turn, losing their characters, form dense areas of wavy, glue-like, interlacing processes, entangling a few nuclei. In the denser portions the muscular fibres of the heart are completely replaced or destroyed, or only appear as small islets of a few isolated fibres; and towards the circumference of any local patch the muscular fibres are observed to present broken or atrophied terminations, and to be more or less widely separated by the intruding tissue. Here and there streaks of pigment granules may mark the site of destroyed muscular tissue.

The process of fibroid infiltration must by no means be regarded as in all cases a destructive lesion; on the contrary, it is in most instances the result of an effort at repair. This is most distinctly the case in heart "scars," where the necrosed muscle, infiltrated with blood elements which constitute an infarct, is gradually removed by absorption and replaced in the only possible way by the growth of a living but inferior tissue, which serves the purpose at least of healing the breach and giving mechanical support to the heart wall. And, rightly regarded, the fibroid infiltration more generally dispersed through the heart substance in cases of retarded or restricted circulation is the means of maintaining the resistance of the ventricle walls to the blood-pressure, a conservative effort, although attended with but poor and temporary success.

In cases of local fibroid infiltration reparative of necrosing infarcts, the scars sometimes become infiltrated with lime salts, and grate under the knife on section.

Symptoms and signs.—The symptoms of general or extensive fibroid infiltration of the heart are those of chronic heart failure, and difficult to distinguish from those of fatty heart, with which, as already observed, the disease is often associated. The patient, usually fifty or upwards, and more commonly a man, has for some months been aware of scantness of breath, and of oppressed feelings about the heart on exertion; but he has become accustomed to this, and the first symptoms compelling his attention, and leading him to seek advice, generally supervene quite suddenly. During some accustomed or slightly increased effort—the walk home from business or an extra round at golf, or a tramp with the gun over a turnip field or up a sharper hill than usual—he is seized with severe breathlessness and oppression at the heart, which compel him to stop and rest for a time and to get home very quietly for fear of a further attack, of which he has some dread. The first attack may amount to a distinct anginal seizure (see "Angina Pectoris," case 3). After this experience his cardiac power is never on the same level as before, and often deteriorates rapidly. His breathing fails him on slight exertion, he becomes liable to dyspnœa on slight distension of the stomach, his face becomes somewhat puffy and dusky in colour, he is apt to be awakened at night with more or less urgent dyspnœa and wheezing, which he regards as asthmatic. The ankles and legs become puffy and œdematous, second sound there is dull, muffled, and prolonged. In mitral cases, however, the second sound over the pulmonary area may be strongly accentuated, although duller and less acute than in the earlier stages of the valve disease.

There is frequently some albumin in the urine, especially in the later stages; and other evidence of visceral congestion from retarded circulation, such as occasional congestion at the base of the lungs, fulness of the liver, and the dyspeptic phenomena of slow digestion with flatulence and loaded urine. With increasing failure of cardiac force the urine falls in amount, and dropsical phenomena set in.

Treatment.—The treatment of fibroid degeneration of the heart is best considered under the diseases—emphysema, angina pectoris, and failing compensations in cardiac lesions, into the symptomatology of which it enters as an important factor.

(δ) *Aneurysm of the heart.*—Aneurysm of the heart is a rare condition, and one still more rarely clinically recognisable. It is questionable whether all the cases recorded by Heschl and Willigk are cases of true aneurysm. The left ventricle is almost exclusively affected and most commonly (in 59 per cent) at the apex; occasionally the septum between the ventricles is the seat of aneurysmal bulging. The pouch varies in size from that of a filbert to that of a large cocoa-nut; it is lined by stretched endocardium, and contains laminated clot and more recent coagulum.

Local destruction of the muscular fibre from any cause may lead to aneurysm. Local softening, consequent on disease or occlusion of a branch of a coronary artery, is commonly responsible for acute aneurysm. Circumscribed suppurative myocarditis is another cause of it. Chronic fibrous myocarditis disposes to aneurysm when the heart wall is thin, not when it thickens.

Dr. Wickham Legg attributes such aneurysms to fibrous degeneration of the heart muscle, and points out that while there is abundant evidence that this degeneration is commonly due to impaired coronary circulation, there are yet many cases of aneurysm of the heart which occur in people under forty years of age, in whom the coronary arteries show no change, so that he doubts whether coronary obstruction is responsible for the myocarditis in all cases. Hilton Fagge regarded fibrous myocarditis as the cause of chronic aneurysm in almost all cases.

The tendency is for the sac to rupture into the pericardium, causing death. In other cases death occurs from mechanical interference by the sac with the movements of the heart. Spontaneous calcification and partial obliteration of the sac may result.

(ii.) **Thrombosis, Embolism,** and (iii.) **Aneurysm of the coronary arteries** require brief notice, although the symptomatology and diagnosis of thrombosis and aneurysm are for the most part included in the phenomena arising from atheroma of the vessels, whilst embolism is a very rare affection, and difficult, if not impossible, to recognise during life.

Embolism of the coronary arteries may occur under any of the conditions which occasion embolism of other systemic vessels; situation of the vessels at the commencement of the aorta, the wi at which they leave the vessel, and the bulk and impetuosity of th current at this portion, are all conditions unfavourable to the pa clot into these small side arteries.

The emboli may be of the ordinary fibrinous character, or, as of ulcerative endocarditis, may be septic. It is quite possible fo from a softening atheroma of the main coronary trunks to be c onwards to occlude some of their terminal branches.

Thrombosis of the coronary arteries is a frequent result of atheromatous change and is also occasioned by specific arteriti portion of the vessel already thickened and narrowed by ather change may thus become more or less suddenly and completely by coagulation. Thrombosis may occur at any portion of the c arteries, but is most frequently met with near their origins fr aorta for the reason that these portions are the most common extensive atheroma. When it occurs deeper in the heart it associated with gummatous arteritis.

It is to be borne in mind that although it has been sh Wickham Legg and West, contrary to the opinion previously amongst pathologists, that there is at least some intercommu between the peripheral distribution of the two coronary vessels, communication is very restricted, and the effect of a complete of one of the coronary arteries in any part of its course is to anæmia of the territory beyond. Fringing the anæmic ar encroaching upon it is a line of congestion or partial capillary stas there is no filling up of the area with blood so as to form the cheese-like appearance of recent infarcts in more vascular tissue yellowish tinge of the area is that natural to anæmic muscle. A s from fatty degeneration and molecular necrosis of the area follo hæmorrhages may occur into the softened area. Microscopica muscular fibres are found broken up, their transverse striæ are l the remains of the fibres have assumed a hyaline or waxy app (Coats). The area of congestion surrounding the infarct becomes of more or less inflammatory reaction, attended with the usual proli of connective tissue, and infiltration with leucocytes. The soften wastes (falling below the surface on section), and gradually un contraction by encroachment of fibroid growth extending fr periphery, the semi-liquefied tissues becoming slowly absorbed; t result being a heart scar of dimensions varying with the size original infarct. In cases, however, where the softened territor considerable dimensions, the branch occluded being large, the s area of the heart wall yields before the blood-pressure, and a aneurysm of the heart is formed which may terminate in rupture.

The result of a partial occlusion of the coronary artery by thr or atheroma has already been described, namely, a fibrous formation of the corresponding territory; and, in cases in wh complete occlusion of the vessel is slowly effected, the same effect is produced.

Symptoms and signs.—The symptoms of sudden occlusion of a considerable branch of the coronary artery generally begin with an anginal paroxysm which may be fatal at once. In cases in which the first seizure is survived, the subsequent phenomena are those of rapid heart failure, dyspnœa with acute anginal paroxysms, rapid and more or less irregular heart's action, dilatation of the organ to the right or left according to the ventricle affected; systemic and pulmonary œdema are also correspondingly predominant. These acute phenomena almost invariably supervene upon chronic heart difficulties already ascribed to degenerative changes, and more or less quickly close the scene. Even the rare cases of embolism of the coronaries have generally been preceded by the signs of acute or chronic endocarditis, usually of the aortic valves.

Aneurysm of the coronary arteries is a disease the secondary effects of which upon the cardiac muscle are of less importance; the disease itself will be treated of in the sixth volume of this work (art. "Aneurysm").

III. IMPAIRMENT DUE TO SENILE CHANGES: PIGMENTARY DEGENERATION; ATROPHY.—(*a*) **Pigmentary degeneration.**—This is a condition seen in nearly all people above the middle period of life, but the change is not met with in the voluntary muscles (Wilks and Moxon). The heart weighs less than normal; it is hard and tough, and the muscle fibres are a dark chocolate colour. The pigment itself consists of hæmatoidin granules of a reddish yellow colour collected about the nuclei of the muscle fibres. Atrophic changes usually accompany the pigmentation, though the striation of the fibres is not much altered. Besides senile states it is met with in any general emaciation (Wilks and Moxon); it does not seem to impair the functions of the organ.

(*b*) **Atrophy of the heart.**—Atrophy of the heart may be part of general wasting, as in old age or chronic disease. It may become reduced in weight—from 9 oz. in woman, or 10 or 11 in man, to 6 or 5 oz., drier in texture from loss of fat and fluid, and darker in colour from the accumulation of pigment granules about the nuclei of the muscular fibres. Local or general atrophy may result from impaired circulation in tortuous and diseased vessels; but under these conditions, except perhaps in old people, the muscular atrophy is attended with the overgrowth of another tissue—the connective. Fatty and fibroid infiltration are both attended with more or less separation and atrophy of the muscle proper.

IV. IMPAIRMENT OF THE HEART FROM FUNCTIONAL STRAIN requires little more than a reference here, since the forms it assumes are discussed elsewhere.

Functional strain, resulting in hypertrophy, may be due to the prolonged endeavour of the heart to overcome some increased resistance to the circulation, or to compensate some defect in its valve mechanism.

In the St. George's Hospital Reports, 1870, and in a previous paper read before the British Medical Association in 1869, Professor Clifford Allbutt describes the effects of overwork and strain on the heart and great blood-vessels, especially to be observed amongst such hard labourers as forgemen, colliers, wharfingers, etc. He also relates some cases illustrative of the earlier stages in which, after excessive exercise in mountain-climbing, hard gymnastics, and rowing respectively, signs of dilatation from acute overstrain are followed by those of hypertrophy of the heart. Professor Allbutt considers the sequence of events to be as follows:—(i.) Dilatation of right heart; (ii.) dilatation of left heart; (iii.) hypertrophy of one or both ventricles; (iv.) chronic inflammatory endarteritis of the aorta; (v.) dilatation of the aorta; (vi.) incompetency of the aortic valves; (vii.) further left ventricle hypertrophy compensating aortic defect; (viii.) degenerative changes ensuing upon hypertrophy. (*Vide* art. "Mechanical Strain," p. 841.)

Mr. Myers in 1870, in a paper on "Diseases of the Heart among Soldiers," drew attention to the effects of prolonged exertion in tight-fitting uniforms, and especially whilst wearing the tight breast-strap, in producing cardiac and aortic diseases from overstrain.

Da Costa has described a condition of "irritable heart" as of very common occurrence in soldiers during the fatigues of a campaign, and observed by him especially amongst the soldiers in the American Civil War. A persistently quick action of heart, with precordial and left shoulder pains, and bouts of severe palpitation under slight exertion, or digestive disorder, are the principal symptoms. At first these are unattended with any notable physical signs, and they may subside without such signs; but in the cases of greater severity or longer duration there is obvious enlargement of the heart. The pathology of these cases would no doubt be for the most part the same as that described by Allbutt, namely, a chronic myocarditis ensuing upon dilatation and mingled with muscular hypertrophy; but probably there is also some direct damage to the cardiac nerves, originating at the terminals of the vagi and sympathetic. (*Vide* Soldier's Heart, p. 851.)

V. IMPAIRMENT OF INFLAMMATORY ORIGIN.—(*a*) **Interstitial myocarditis.**—Myocarditis most generally consists of an irritative overgrowth of the interstitial connective tissues of the heart, which may extend from a pericarditis or an endocarditis. In its first stages an increased nuclear proliferation, permeating the muscular fibres, causes a "cloudy swelling" of the tissue, and a certain degree of increased softness to the touch, but the later result is more or less fibrous toughness of the part involved.

Disturbed circulation, general or local, will occasion cardiac fibrosis; such as chronic congestion of the cardiac veins, or restricted or obstructed circulation through the coronary vessels. The cicatricial or tendinous patches of the heart are produced by interstitial myocarditis. An impairment even to destruction of the true muscular fibres of the

heart necessarily ensues upon local or general fibrous my
Dr. Charlewood Turner has pointed out that interstitial my
may exist and extend apart from any affection of the perica
endocardium, and that in cases of dilatation of the heart o
hypertrophy, from whatever cause, this morbid process is at w
responsible for further changes; lastly, Dr. Turner points
the occurrence of recent exudative and older fibroid chang
together in cases of valvular disease and secondary to Bright's
indicate the one change as being the initial stage of the other.

(b) **Parenchymatous myocarditis,** which is met with in cert
of septic poisoning, such as pyæmia and diphtheria, is prob
a very active form of the preceding process in its earliest
Professor Leyden has studied this lesion most carefully, and
it as an acute myocarditis characterised by intermuscular
proliferation and by secondary atrophic changes towards necr
deposition of pigment; fatty degeneration of the muscular fibre
panies it, possibly in consequence of the inflammatory change
form of myocarditis is always secondary to infective fevers,
diphtheria, scarlet fever, and the like; and has been met with in
or less degree in all acute febrile diseases, rheumatism, cereb
meningitis, variola, erysipelas, malaria, septicæmia, influenza,
forth. In enteric fever and in gonorrhœa the respective speci
and cocci have been found in the heart.

(c) **Purulent myocarditis** is in most cases secondary to
embolism of the coronary vessels; as in pyæmia, and ulcerati
carditis. In all cases infective microbes are conveyed to the
muscle through the coronary arteries, and set up foci of
myocarditis resulting in minute or larger suppurations.

(d) **Syphilitic myocarditis.**—Syphilitic myocarditis almost in
occurs either in the immediate neighbourhood of a gumma or s
to and in the territory commanded by a specific arteritis. A
was first drawn to the occurrence of syphilitic lesions of the my
by Sir Samuel Wilks in 1856; and many isolated cases ha
reported since at the Pathological Society of London and in
English and foreign medical journals. Our knowledge of the
however, is mainly derived from the post-mortem observation
in patients, by no means all of whom died with heart symptoms.

Syphilitic disease affects the myocardium in one of three w
either singly or combined:—

(α) There may be syphilitic arteritis and secondary or c
chronic myocarditis.

(β) There may be gummatous formation in the heart wall, aro
extending from which chronic myocarditis takes place.

(γ) There may be a diffused chronic myocarditis of specifi
affecting a considerable portion of the heart. It is doubtful,
whether this latter form of diffused syphilitic myocarditis
originate in scattered gummatous depositions.

Syphilitic arteritis of the coronary vessels does not differ from similar arteritis elsewhere. The endocardium is only affected in cases in which there is subjacent gumma of the muscle, and the pericardium as a rule also only in connection with such gummata or gummatous affections of the vessels. Pericardial adhesions in syphilitic subjects are, however, not uncommon. Syphilitic disease, limited to the valves of the heart, is almost unknown, but the aortic valves frequently partake with the aorta in an endarteritic thickening having its origin in the syphilitic cachexia.

With the undue rigidity, actual narrowing and occasional thrombosis, with which coronary arterial diseases of syphilitic source are attended, we find fibrous degeneration, dilatation, angina pectoris, and so forth, as later consequences.

Gummatous formations may occur in any part of the heart, most commonly in the ventricles or septum. They have the usual features and ill-defined microscopic characters of gummata elsewhere; they may soften, or undergo fibroid change, and they are always surrounded by more or less spreading fibroid condensation of the heart wall from associated chronic myocarditis.

It cannot be said that any symptoms have yet been formulated which in their grouping or individual significance are characteristic of syphilitic disease; and for the obvious reason that very different portions of the heart may be affected in different cases, and that the upshot of the morbid condition in each case is a spoiling of the cardiac muscle at the part affected, and more or less interruption or spreading disturbance of the cardiac mechanism therefrom.

Functional irregularity, anginal seizures, syncopal attacks, any of which may prove fatal, are amongst the most common symptoms. It is remarkable that sudden death has terminated a large proportion of the recorded cases of gumma of the heart in most instances without any previous recognition of the disease. The first case recorded by Sir Samuel Wilks ended in death in this way, as did fourteen out of twenty-five cases recently collected by Dr. S. Phillips. Enlargement of the heart, or displacement of the apex beat to the left, or more marked evidence of enlargement to the right, are amongst the later signs; especially in cases of the more diffused form of syphilitic myocarditis.

The absence from the history of the case of rheumatism, of gout, of alcoholism, or strain; and evidence—whether in the form of a distinct history or of collateral lesions of a specific kind—pointing to a syphilitic cachexia, are circumstances which, in the presence of such signs and symptoms of cardiac disease, may lead us to suspect its syphilitic nature, and to direct our treatment to that probability. When the signs and reservations with regard to such heart diseases as are above spoken of are found in men, and before middle life (nicotine poisoning being also excluded), an additional argument in favour of syphilis will be found. The success of antisyphilitic measures of treatment, which, however, would be combined with cardiac tonics, strychnia, digitalis,

iron, or arsenic appropriate to the case, would further hel
diagnosis.

VI. TUMOURS OF THE MYOCARDIUM.—The heart is one o
organs least commonly affected by new growths. Primary growt
exceedingly rare, but sarcoma, myxoma, fibroma, carcinoma, and
interstitial tumours have been met with. Tubercle, common i
pericardium, is rare in the heart substance, and then occurs a
exclusively as an accompaniment of general tuberculosis. Moreove
heart is but rarely invaded by secondary growths. Round-celled sa
is occasionally to be met with, extending apron-like over the perica
greatly thickening it, and embedding the great vessels, yet not inv
the heart itself. Carcinoma of the lung and mediastina frequ
invades the parietal pericardium in cauliflower-like excrescences,
yet spares the heart. Sarcoma sometimes invades the heart fro
mediastina along the sheaths of the coronary vessels and their ran
tions, penetrating into the intermuscular tissue and separating the c
fibres, causing them to atrophy (Boyce). Secondary cancerous de
both epithelial and medullary, have been met with in the substa
the heart at post-mortem examinations.

VII. PARASITES.—Hydatid is rarely met with in the heart o
human subject, but it does occur, as does also cysticercus cell
The cysticercus of tænia solium is common in the heart of swine
that of tænia mediocanellata in cattle; but they are rare in man.

Actinomycosis may extend to the heart from the mediastina
lungs.

Trichina spiralis, according to Wilks and Moxon, is never fou
the heart, or extremely rarely.

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DISEASE OF THE AORTIC AREA OF THE HEART

In formal presentation of this subject it is customary to divide it into two parts—into aortic stenosis and aortic regurgitation. To carry out this division, however, leads to some embarrassment: on the one hand, by far the larger number of cases of aortic regurgitation are attended with signs of interference with the issue of blood from the left ventricle; on the other hand, cases of stenosis of the aortic orifice may be attended with regurgitation. Again, the causation of the two evils is similar if not identical, and the determination of the one or the other event may be accidental; so that, although the clinical features of the two events, taken singly, are very different, as the two are often coincident it seems more convenient to take them together except in those sections in which their distinction becomes imperative.

Subject.—By aortic regurgitation we mean that in diastole some of the blood driven into the aorta returns to the left ventricle; when we hear the sound characteristic of this disorder the inference that the aortic valve is out of order is almost irresistible. A definite diastolic murmur heard in the areas of the murmur of aortic regurgitation is perhaps the surest diagnostic indication of its kind. Not so with aortic systolic murmurs: of such signs these are perhaps the least definite. I need not say that an "aortic systolic murmur" may not be significant of organic disease at all; or if significant of disease about this orifice the alleged stenosis may be more apparent than real, the murmur may signify no more than a roughness or other deformity of the part implying no constriction of the orifice; nay, it may be consistent with dilatation of the orifice. Clinical clerks are far too ready to assume aortic stenosis in all cases of organic disease of this orifice revealed by a direct murmur; aortic obstruction, though open to some objection, is a better name.

instance, as the concurrence of aortic disease in women engaged in labours harder than those usual in the sex—in women who have worked in the fields, in washerwomen, in women employed in brick-making, or on the bank-tops of mines. Making every allowance in such cases for the greater exposure to weather, there seems to be a greater prevalence of aortic mischief in such women after acute rheumatism than among women who have led lives of less muscular stress. I have not found that either in alcohol or syphilis we have factors to invalidate these propositions; but to pursue them much farther would be to trench upon the subject of endocarditis entrusted to abler hands than mine: suffice it to say that I conceive that although in an unusually severe attack of acute rheumatism both sets of valves may be attacked, whether directly in each area or by extension from the mitral to the aortic, yet in ordinary attacks, if the aortic valve be involved as well as the mitral, it will often appear that the patient, either in work or play, has been wont to put out considerable muscular exertion. Disease of the aortic valve alone is a most unusual event in the young subjects of chorea (of 250 cases Gowers found aortic regurgitation in two, and obstruction in one), whose muscular efforts are fitful, not exacting; indeed its association with mitral disease in this disease is rare enough.

The predominance of rheumatic inflammation on the left side of the heart is often explained likewise by the fact that mechanical stress falls more hardly on these valves than on those of the right side, and so it may be; yet it is not easy thus to explain this preference: are we to assume that muscular labour in these persons had already produced some cardiac strain, and that before the rheumatic attack these structures were more or less impaired? This would seem to be a grave charge against the physical uses of the body; a charge which on the face of it seems unreasonable, if so be that without the rheumatism no harm would have come of them. A remote suspicion of such a deterioration can scarcely justify us in discouraging all exercises beyond nursery games. Short of lesion one would anticipate that increased work would enhance nutrition, and thus fend off rather than invite the approach of disease. Roy's article on the elastic properties of the arterial wall may, however, be usefully consulted on this problem.

Syphilis is probably concerned in the causation of many cases of aortic disease, though, except when it exists in the form of a definite gumma, we have no certain test of the syphilitic process, whether in the living or in the dead body (p. 905). How large a factor, however, syphilis may be in arterial disease will be shown by Dr. Mott in the article on this subject in the next volume. Dr. Parkes Weber (95) finds that syphilis is apt to be the starting-point of atheroma. For many years I have been wont to infer from the state of the radial artery the effects of syphilis on the vessels of almost every man who had been saturated with this poison; and such surmises have been reinforced by the more direct observations of Dr. George Oliver. We can scarcely suppose that a destructive agency, so active as we know it to be in all other arterial regions, should be without effect in the aortic area of the heart; yet in deciding in a particular case t aortic lesion is syphilitic, we are confined to the inferences which drawn from the story of the case or from associated changes elsew which indications may, indeed, bring us to a moral certainty. W that a comparatively young man of otherwise healthy habit does no from local disease of the aortic region of the heart unless it be in quence of extraordinary muscular stress, of rheumatism, or of syphi that although there may be no direct means of detecting the s yet if muscular stress and rheumatism be both denied, we fal upon syphilis as we do with some assurance in the case of aneurysm in such a person; the inference, pathologically sp may not be positive, but it is usually justified in practice. The fol case illustrates these remarks (*vide* also art. "Tabes Dorsalis, following volume):—

Dr. Pye-Smith reported a case of a man, aged 32, who died wit disease, the physical signs being those of aortic obstruction and regurg Rheumatism and chorea were excluded. Atheroma was improbable o the comparatively young age of the patient, who was, moreover, not su laborious work. After death there were no signs of rheumatic or i endocarditis, but a patch of recent aortitis and deformity of the valve lesion was soft, injected, with a swollen, crescentic margin suggesti advancing edge of a secondary syphilitic eruption of the skin; there atheroma. The only other evidence of syphilis was a fibroid condition testicles, though this was not very marked. He suggested that the sy aortitis had spread to the valve and so produced the disease in question (

Atheroma.—A full discussion of the nature and fashion of this or chapter of diseases of the arteries is deferred to the next v Here we may ask whether the disease of the aortic orifice sheds an on the origin of this insidious and rather peculiarly human d Does it appear that muscular labour plays any important part origin or determination of the change? For my own part I cann that, likely as it may seem at first sight, there is much evidence in of this hypothesis. It is true that this disease also is found m less exclusively on the left side of the heart—the side of stress also true that atheroma may be the ultimate form of arter whatsoever origin—rheumatic, syphilitic, or mechanical—mechanica the pulmonary artery in mitral stenosis; still we must admi atheroma is as likely to occur in the elderly lady who has spent h in trotting amiably about the parish, as in her husband who has for his falls, felled his own trees, and stumped about after his bird his boyhood.

Again, atheroma is by no means constant or approximately u in its position: although well marked, no doubt, on the greater cur of the arch where tensile strain is highest, and at bifurcations a flexions, yet it does not by any means confine itself to the parts receive the main stress of muscular exercise, or to parts where, tissues being most abundant, tone is least and tension most. On t trary, it is one of the surprises of practice to find it in all sorts of odd areas; and within such areas it is patchy. If in one necropsy the cerebral vessels are like branched corals, in another, with atheroma enough elsewhere, the cerebral vessels seem clean enough. In one body atheroma is abundant about the region of the heart; in another, the heart and its orifices are fairly normal, but extensive patches of atheroma are discovered in the abdominal aorta or in peripheral areas of the arterial tree. Such contrasts are too well known to need the support of recorded cases in this place.

Again, is it that the main cause of atheroma of the heart is mechanical stress, yet stress due not to muscular exercise, but to that more persistent high arterial pressure of constitutional origin which may be established as well in the squire's wife, with her indolent habits and gouty inheritance, as in the sportsman himself who works off his meat and drink day by day in the fresh air? Is the comparative freedom from atheroma enjoyed by animals to be attributed to the fact that they do not suffer from chronic high arterial pressure; that they have exercise enough—muscular stress enough, many of them—but are fed by their owners, and fed therefore economically? Certainly we see daily that hard exercise keeps the ill effects of a too vigorous appetite at bay. This is clinical gossip, I fear, rather than science; but we cannot at present get much nearer to the facts. Frequent high blood-pressure, then, as in excessive muscular stress, and more persistent high pressure due to luxus-consumption relative or positive, to gout (especially in its non-articular forms, for the frank articular form of gout leads less surely to high arterial pressure), to lead poisoning (by way of gout), and possibly to certain products of metabolism engendered in old and defective organs or tissues, may produce atheromatous changes which often involve the aortic region of the heart, directly by friction and local irritation as in central and distal arteries, or indirectly by more immediate mechanical strain as in more central arteries. To quote Dr. Balfour, "there is a consensus of opinion that the arterial system is that upon which the finger of decay is first laid." We see daily in the post-mortem room, yet still with some surprise, how readily the heart even of an old man may take upon itself no puny hypertrophy. It is no unusual thing to find a big heart, and one big with no bad stuff, in old persons subjected in later life to increased blood-pressure, even when the coronary arteries have undergone some measure of deterioration; in such cases the aortic valves, even if competent, are practically always thickened. Still, with all this, can we say that aortic regurgitation, common as the disease is, is frequently found in the decay of elderly persons? I think not; on the other hand, it seems much less common in them than aortic obstruction—by which I mean no more than the presence of an organic direct murmur; now an aortic systolic murmur may continue as long as life holds together, and afford one of the many evidences of the long story of cardio-arterial degeneration. This form of aortic disease is rarely of itself the immediate or proximate cause of death; we may call it but an accident in the course of a general cardio-vascular involution, of some other cause of arterial degeneration, while, at the same time, he has been following a laborious employment: yet we may fairly presume, perhaps, that in many such cases muscular stress and decay together have gradually impaired the valve to the point of insufficiency. As I have said, senile decay alone does not usually cause aortic regurgitation; more commonly it produces disease of the aorta with implication of the orifice, which is betrayed by a direct murmur. When, therefore, we find that regurgitation occurs rather in persons under fifty years of age, of the laborious sex, and especially in men who have been engaged in heavy toil, we cannot escape the suspicion that muscular stress, if not the sole or always the chief agent in these cases, is at any rate a potent determining cause. Acquired aortic disease in children is one of the rarest of clinical cases; even in the acute rheumatism of women and children we have noted that the mitral valve is affected first, and that if the aortic be involved it is as it were by overflow (p. 908).

Once more, if not infrequently, yet almost exclusively in men, we discover that aortic insufficiency establishes itself in patients under the age of senile degenerations, free from evidence of syphilis or other infection, including rheumatism, and telling no story of sudden rupture, shall we not be justified, at any rate in the large majority of instances, if we suppose that the disease may be attributed to the accumulated effects of muscular strains recurring at longer or shorter intervals over a number of years? Finally, if a man of irregular habits, and deteriorated tissues, describes to us the symptoms of sudden rupture of the aortic valve, we may reasonably infer that an effort, inadequate to rupture a healthy aortic valve, had sufficed to rupture a valve already impaired.

Thus, in the causation of aortic insufficiency due to muscular strain, we are led to recognise three classes, namely, acute rupture; chronic forcing of a valve previously impaired by some constitutional poison, such as syphilis; and chronic forcing of the valve by the importunity of repeated strains none of which alone was sufficient to break down a healthy valve, but all of which, by molecular rather than massive strain, contributed gradually to increase valvulitis and to break down the resistance of the part. The condition of the tricuspid valve in protracted cases of mitral stenosis is an excellent example of chronic valvulitis due to stress. As in the case of the housemaid mentioned in the last paragraph but one, this strain of the tricuspid tends to stenosis. We may note in passing that, in respect of prognosis, it is important to know whether and for how long the ruptured valvular limb is supported by tissues otherwise healthy. Although I have said (p. 910) that atheroma as a general disease of the arterial tree is not due, in the main, to muscular stress, yet local atheroma very often has this origin; it is indeed the common result of more than one kind of chronic arteritis.

It has been alleged that prolonged acceleration of the heart, as in Graves' disease, may produce the valvulitis of strain; but unless the sum of work done be considerably increased, which is not usually the case, such a result is not to be anticipated.

External violence.—Finally, one or more of the aortic crescents ruptured by a blow on the outside of the chest. Within my o perience blows or crushes resulting in the split of a vessel af manner have produced aortic aneurysm, not rupture of an aortic still there are many cases of this kind on record. I distinctly ren indeed, in the Leeds Infirmary a case of mitral stenosis which, af closest inquiry, we confidently attributed to the kick of a horse cardiac area; the patient was a young man, and the symptom long in declaring themselves; yet the connection between ant and consequence seemed inevitable (p. 864). Dr. L. Heidenhain of wald has studied these cases; his conclusions are that, with or injury to the ribs or obvious external bruising, an external blow rupture a valve in the heart, may (*b*) damage or rupture the muscle, or rarely (*c*) set up a stenosis by insidious endocarditi Samuel Wilks has recorded a case of this kind which occurred in aged nineteen: a blow on the chest ruptured the posterior cusp aortic valve from its free margin to its base. A small deposit o had begun to form on the raw edge. An analogous case, in wh heart itself was ruptured in a lad of sixteen, by a blow on the ches caused no external bruise, has been reported by Dr. William G Wisbech, and the preparation is now at Cambridge. Potain argu if a blow, such as a jockey received who was heavily thrown so chest smote the ground, rupture an aortic cusp, the heart at the of the blow was in systole and the aorta distended. In cases of ru the mitral valve in like manner, of which he records two, he concei heart to have been in diastole when the blow fell, and the ventric

Nervous shock.—In the earlier medical writers, not in the poets often meet with the assumption that intense emotion may be attend injury to the heart. In any careful consideration of this point we divide the question: we should first consider injury due to inte with the circulation itself more or less directly; as, for example, an effort of the inspiration as to force the intra-thoracic negative p to an extreme; and, secondly, indirect interference through the system. Of the first kind of case I remember a strange exa the West Riding Asylum at Wakefield. A woman, afflict violent mania, one day in a fury held her breath preparatory to burst; she became livid, fell to the ground, and died (93, p. 14 the necropsy it appeared that death was due to extreme fuln dilatation of the right heart and venæ cavæ, though it is possible was due to a fulminating shock, by way of the vagus nerve, arrest auricles. Of death through the heart, clearly dependent upon shock alone, I have no experience. All that we know, as yet, res nervous influence on the mechanics of the heart, is that vagus sh diminishing auricular contractions lessens the output of the heart is also slowed, and its diastole enlarged. This for the heart is servative function, but it is conceivable that, even in a healthy adult, be carried too far. The depressor effect, produced through dil of the splanchnic veins, could scarcely harm the heart. The accelerator nerves are probably stimulated during emotion, which, as we all know, quickens the rate of the heart; but if, as we may presume, the output is proportionally less, and the resistance less rather than more (if the depressor be influenced also), no excessive mechanical strain thus falls on the organ. Augmentor action is too little understood to allow us to argue conclusively about it. Intense emotion might be attended with a universal or very widespread constriction of the peripheral vessels by which blood-pressure might perhaps be dangerously raised; if such a constriction occurs, however, it is transient, and relaxation of these vessels and of the sphincters seems to be the ordinary effect. Moreover, we see no great cardiac distress during a rigor, or after many returns of quotidian ague. So far as experiment goes, it would seem indeed that nervous shock tends to lower the blood-pressure. Finally, it is a reasonable surmise that some deterioration of the nerves or their centres, due to prolonged mental distress, might be followed by fatty degeneration of the cardiac muscle; such a process scarcely comes under the head of aortic strain, and it is at least as likely that such impairments of its nutrition take place by way of the blood. What may be the truth concerning these problems, however true it may be that prolonged grief may invalidate the chambers of the heart, such evidence as we have, physiological and clinical, seems to indicate that the aortic machinery at any rate is subjected to no especial stress, but, perhaps, rather the reverse.

Pathogeny and Morbid anatomy.—Whether the heart be liable to undergo primary hypertrophy under normal or relatively normal conditions is one of the most important problems which meet us at the outset of this section. The answer to the question is not yet given; but the opinion that it is so liable is not without strong support (p. 903): so far, indeed, as my reading goes, I think that the affirmative opinion is gaining ground. For my own part, I find that to be assured of the lesser degrees of hypertrophy of the left ventricle is a much more difficult clinical task than I used to suppose; unless, of course, the hypertrophy be attended with notable dilatation, and a clinical dilatation would surely forbid us to describe the conditions even as quasi-normal. Unless the person under observation be an inordinate drinker of fluids, alcoholic or other (p. 902), there seems no reason to anticipate increase of the mean ventricular output; if, however, the sum of the conditions of resistance is higher in amount than under ordinary circumstances, the supervention of hypertrophy may be explicable; and in this case distension might no more take place than under the fluctuations of output in persons leading a life in which muscular stress is not an important condition. Prolonged exertions in untrained men make themselves felt, as we have seen (p. 849), by more or less uncompensated dilatation; but perhaps in men such as sprint runners, putters of weights, wrestlers, and the like, in whom sudden and repeated efforts, under which initial rises of pressure are frequent, bear a large proportion to more regular exercises, the mean blood-

pressure may rise, as the maxima are high and of very frequent rec
If this be so, simple hypertrophy may follow, though I find such
very difficult to verify. However, healthy men do not come to the
and in unhealthy men the conditions no longer apply. In slin
chested youths with wide costal interspaces a thumping or un
heart may well be mistaken for a hypertrophy; and a great many
men have rather thudding hearts. Whatever books may repeat,
easy task to appreciate a moderate hypertrophy of the left vent
many are the sources of error, as for instance in the relation of
the cardiac area, in chests of different build. Violence of impuls
no means directly related to the volume of the heart or to the
pressure; the "heave" in the impulse, a quality not insignifican
the hypertrophy is considerable, may be hard to appreciate in the
of hypertrophy we are now contemplating; and a slight vertical d
ment of the apex is no less difficult to ascertain, seeing that the f
the chest and its landmarks are far from constant. The resear
Myers, Da Costa, Thurn, Fraentzel (29), and others, on hypertr
the heart found in men submitted to physical stress, were made
upon soldiers (*vide* p. 851). In these men, however, contingent con
have to be considered: omitting drink and syphilis, many ill-f
trained, half-developed recruits are (or then were) clad in ill
clothes, girthed with belts and breast-straps, loaded with 20 lbs. an
of weapon and kit, and unavoidably sent on long harassing marc
which they are untrained. In civil life we see the muscular or
muscular evils which flow from like causes, and we see how tedio
be the recovery from them. Now, if we turn to sailors, to whom
and syphilis are not unknown, but who are clad in easy dress a
"trashed about," we hear nothing of cardiac hypertrophy.

It is said that in hard-worked animals, such as greyhounds an
horses, simple hypertrophy of the heart unassociated with cardio-v
disease is met with. I have referred this question to Professor M'F
of the Royal Veterinary College, who replies, "I have not form
opinion that an amount of muscular tissue notably above the a
is ever found in the heart of the horse or dog as the result o
muscular stress, but that hypertrophy of the left heart is alwa
result of some morbid condition of the valves or of the arteries.
muscular effort were a cause of simple cardiac hypertrophy it sho
almost the rule in bus horses, and such is certainly not the
Arguments from analogy must not have much weight until ve
and we must regard hypertrophy of a hollow viscus in a differen
from that of a solid muscle such as the biceps; moreover, we h
the heart not only a hollow organ, but a hollow organ in in
activity; finally we do not know that the heart of a sound man e
in active pursuits is over-worked on the whole, seeing that the mac
for the equilibration of arterial blood-pressures is of miraculous eff
(*vide* p. 472 and p. 846).

The load factor of the heart, the ratio between its average maximum work, is ample; as Cohnheim impressed upon us, the heart has a large "reserve capacity." If, say, by partial ligation of the pulmonary artery the resistance be increased to three or even four times the normal mean, the arterial blood-pressure will remain constant, although the left ventricle may be doing three or four times its ordinary work. I must not delay any longer on matter which is dealt with in other sections, but these inquiries are germane to my subject, as we are inquiring first whether hypertrophy of the left ventricle is a quasi-normal consequence of muscular exertion, and, if so, whether it can of itself provoke aortic disease, in the adjoining section of the aorta or at the orifice itself. If excessive pressure is thus induced we may see how muscular stress may lead to aortic disease. Roy and Adami noted (*vide* vol. i. p. 123) that "when the aorta of a dog is suddenly and greatly constricted, and consequently the pressure in the proximal portion of the vessel greatly increased, the plasma of the blood is forced into the cusps of the aortic valves, and vesicles of lymph make their appearance on the under surface in that region where fibroid thickening is most frequent in cases of chronic high arterial pressure." This is probably the way in which the chronic inflammation of the tricuspid valve is established which not infrequently ensues upon mitral stenosis. Our study of muscular exertion, however, as we have seen, suggested to us that such work does not create a state of abiding high pressure, but of intermittent high pressures more or less compensated by a mean pressure habitually rather low.

Are we not led, then, to suspect that muscular exertion, unless very sudden and excessive, and attended perhaps by fixation of the chest walls, arrest of breathing, and by some phase of differential pressures in ventricle and aorta of which we know little—in which case we know that a valve may be directly ruptured—needs some other factor to bring about aortic disease? This factor may be one of the causes of constitutional high blood-pressure; or, on the other hand, it may be some debilitating cause, such as syphilis, anæmia, or "misère," which so reduces the normal strength of the cardio-vascular textures that ordinary blood-pressures are high relatively to their feeble durability. A patient of mine, who had certainly done all that he could to strain his heart, if by physical stress it could be done, died of dilatation ("true aneurysm") of the aorta, a result put down unhesitatingly by his friends to over-exertion; yet I knew well, what no one else knew, that there had been an old syphilitic infection, and that not a few evidences of the infection, among which had been symptoms of encephalic arteritis, had from time to time betrayed its persistency. To my mind it was far from clear that muscular stress had the first place in dilating the great vessel, although, no doubt, the vessel once disintegrated by arteritis, muscular stress accelerated the evil. If we are to form a definite opinion of the part played by muscular stress in the causation of aortic disease with or without the intermediation of hypertrophy of the left ventricle, we must weigh with it in the particular case all other factors which may have conspired to the same end. If we except active destruction such as that of infective or severe

rheumatic endocarditis, it may be true that all aortic disease is d
or less to the effects of arterial blood-pressure.

Peter, Traube, and others have insisted upon a distinction l
aortic disease originating in the heart itself, such as that of rh
valvulitis in young and otherwise healthy persons,—cases in wh
cardiac affection is in its initiation a local disease,—and implic
the heart in a more general constitutional change such as syp
arterio-sclerosis—wherein the heart disease is but an expressi
general disease. These divisions have been distinguished b
names as "cardiopathy" and "arteriopathy." The distinction i
making, though it has been made far too literary; in it to
heed has been taken of the tendency of cases in practice t
these logical devices; and much harm is done, especially by
writers, in decorating the several phases of a continuous
tion by imposing names which suggest differences in ki
at any rate in quality, which do not exist. While
cardiac disease tends to generalise itself, constitutional disease
much of its peril from the cardiac factor; the series, starting
true, from opposite points, meet and overlap; thus, un
be in well-marked extremes in the interpretation of which
not likely to go astray, the distinction is often too artificial
of much service. Still, on paper at any rate, the contrast i
making as a study of origins, for reasons which have already ap
and occasionally it may influence the prognosis and treatmen
particular case. The observations of Roy and Adami, already
throw some light on the process by which blood-pressures, re
excessive, set up that opacity and condensation of the valves w
see well marked in the aortic valve, and clearly enough at times i
valves also, under high blood-pressures; as the heart suffers so n
aorta (74), and atheroma may invade no small part of the intra-
arterial structures. Whether, then, the initial injury be such as th
be a rheumatic valvulitis or a syphilitic, the valvular lesions ma
into a common form which we conveniently call atheroma; and, t
between the aortic area of the heart and the aorta itself being no
all, we find this atheromatous change not only in the valve, but spre
often widely spread, in the neighbouring great vessel also: thus th
dilates, its elasticity is impaired, its walls are diseased, and the
caught in a vicious circle, has to meet an increased resistance. Fo
account of atheroma the reader is referred to the article on "Dis
the Arteries" in the next volume; I may briefly say of the valves t
milky opacity of the acuter stages of valvulitis is followed by an i
of fibrous tissue, both in the fibrous ring itself, where it becomes
sive, and in the valves themselves, chiefly about their points of
contact and the corpora Arantii. With the deformities secondary
contraction of these cicatrised tissues we are but too familiar: ind
of the fibrous ring, or of the infundibulum below it, leads to st
and of the limbs of the valve to their contraction, puckering, or oc

Thus the valve may become incompetent, or the orifice contracted; or these results may be concurrent.

The following remarkable case of sub-valvular constriction is published by Dr. Langwill. The patient was a poorly-developed lad of nineteen. He complained of pains in the chest on exertion, though he worked as a foundry labourer till four weeks before admission. A strong, systolic thrill was felt at the base, and a loud systolic murmur was audible five inches from the chest. The chief cardiac disease found at the necropsy was as follows:—

Pathological Report on Heart, by Dr. Shennan, Pathologist to Leith Hospital.

Right auricle.—Normal; tricuspid orifice, 1·2 in. *Right ventricle.*—Nothing particular to note. *Left auricle.*—Endocardium somewhat thickened. *Mitral valve.*—Cusps slightly thickened, particularly inner cusp. Musculi papillares small, and fibrous at apex where they join the chordæ tendineæ. *Left ventricle.*—Walls hypertrophied; cavity 3 in. long; thickness of walls varies from 1 in. to 0·5 in. There are a few narrow fibrous bands stretching across the cavity, at whose points of attachment to the wall there is marked thickening of the endocardium from old endocarditis. On passing the finger up towards aorta, it passes through a fibrous ring—0·7 in. diameter—about 1 in. below the aortic opening. This is continued on to the ventricular surface of the inner mitral cusp. In this position, and extending upwards from the ring on to the lower surface of the postero-external aortic cusp, is a narrow band of comparatively recent vegetations. These cover the lower surface of all the aortic cusps, which also show fibrous thickening and contraction—cone diameter of the opening being 0·7 in. Above the valves the aorta dilates slightly—1·2 in.—but in the second half of the transverse part of the arch begins to contract, so that at the upper part of the descending aorta the cone diameter is 0·6 in.

Thickening of a limb of the valve, says Prof. Hamilton, may lead to the formation of a relatively large spur which, by intruding into the area of the two other shrivelled cusps, may accidentally prevent regurgitation.

From such rough edges and points "vegetations" may sprout, and form fringes on the free or ventricular edges of the valve, rarely on its aortic aspect, whereby friction is increased and extended; and chronic inflammatory changes operate on the endocardium, where the diseased valve brushes it, on the valvular structures themselves, and on the corresponding aortic surfaces. Below the valve "ripple-marked" thickening of the endocardium, due to the strain of aortic regurgitation, has been demonstrated by Dr. Glynn and other observers. Hamilton reminds us that, in disease of the aortic valve, it is rather the base of it which is the seat of the mischief, while the cusps may even be free; in the mitral it is the edge of the cusps and their substance which suffer first. Conversely the valve may be very incompetent, while the orifice is as wide or wider than its normal section.

Stenosis depends often, but not always, on contraction of the fibrous ring: not always, for adhesion and condensation of the limbs of

the valve may narrow the orifice, converting it into a slit o
as is so well known in the case of the mitral valve. In a ca
recently occurred in Addenbrooke's Hospital, under the care of
Bradbury, the adherent margins were united along their su
apposition, and the blood seems to have been forced through
chink so small as almost to evade discovery even on inspection.
was no regurgitant murmur during life, nor was any regurgita
tected by Professor Kanthack. In this case calcification was far
in the ring, no doubt, as well as in the valve; indeed, in steno
buted to the valves the ring is usually concerned in the misch
Similar cases have been published by other physicians.

I need scarcely say that the presence of "vegetations" and
detachable fringes on these dog-eared cusps is a matter of far m
local importance, as by their means embolism may come about.

Ulceration of one or more of the limbs of the valve is always
ous process. When dependent upon micro-organisms, and we ca
how frequently they enter in, the process may be terribly destru
the records of infective endocarditis give us too much reason t
on the other hand, decay or perforation may be very gradual,
always due to infection: probably in chronically diseased valves
be a mere mechanical disintegration. Perforation of a seg
said to betray itself by a piping quality of the regurgitant
Other rasping or "musical" qualities of these murmurs are attrib
the projection of spurs or shreds of segments which, fluttering o
ing as reeds, give peculiar qualities to the sounds. It is commo
that murmurs may be generated by a rough surface, as a brook
over pebbles; this assertion must be taken with considerable
for to produce a murmur the column of the blood must be broke
a merely mammillated or corrugated surface will not do
the eminences be such as to set up vortices around or behin
The common notion that murmurs may be generated in a roug
without any contribution from the valves or orifice, is improba
not supported by experience. If, in the absence of any cause in th
lar area, such murmurs arise, which is rather doubtful, they ar
attributed to dilatation of the aorta, wherein vortices may form
the slower external and the swifter internal layers of the issuin
However, we meet with cases every day of advanced disease of t
in which no systolic murmur is heard. Again, that there is mo
the satisfaction of an anatomical curiosity in the endeavour to fix
competence or the obstruction upon this limb of the valve or th
indisposed to believe; nor can a leaf or stump of a diseased segment
the access of blood to a coronary artery, unless, of course, it so ad
the wall of the aorta, or the mischief so extend from it, as to ch
mouth of the vessel. That the propagation of a regurgitant mu
this direction or that can indicate the limb of the valve affected, o
affected, is not very probable in itself, nor is it borne out by exp
and that the deformation of one particular limb of the valve shoul

the coronary circulation more than the same incompetence in another is impossible; there cannot be differential pressures within this area. Congenital peculiarities of the aortic valve and coarctations of a congenital origin are dealt with in the article devoted to this subject (p. 706). In simple rupture of a healthy valve one limb only is torn in the large majority of cases; but a few cases of the rupture of two limbs are on record. Generally the limb is rent on the free edge, but sometimes it is torn from its attachment to the vessel.

Whether these chronic changes in and about the aortic orifice lead to regurgitation or to stenosis without incompetency, crucial as the distinction is in clinical medicine, is pathologically a matter of accident; the result depends, that is, on contingent causes. At the same time it is well to remind the pathologist that to test the competency of an aortic valve by means of a column of water, a test which is more useful in the post-mortem room than one might expect, is insufficient in a doubtful case, unless the height of the column of water be equal to the maximum aortic pressure—to the pressure, say, at the moment of aortic diastole of 180 mm. Hg. Moreover, the water may even then escape from the coronary arteries. Practically, as the water brings the valves into apposition we have to judge of their competency by the eye.

Of "gouty valvulitis," of a primary kind, after the manner of rheumatic valvulitis, and apart from the chronic sub-inflammatory and degenerative changes in the aorta resulting from abnormally high arterial pressure, I have no knowledge, either pathological or clinical.

It is very important to remember that these degenerative changes involve the area and, sooner or later, the orifices of the coronary arteries; so that the heart, instead of enjoying that increase of nutrition which its greater work demands, and which at first the hypertrophied ventricle supplies, may receive, after the first stage of the malady, considerably less than its normal nourishment.

It is commonly asserted that insufficiency of the aortic valve may come about, not from any defect in its own structure, but from dilatation of the aorta, whereby the sectional area of the orifice is enlarged. Intermittent or temporary aortic regurgitant murmurs are thus explained. Barié, a careful and experienced observer, reported thirteen cases of aortic regurgitation from widening of the orifice without disease of the valves; but a persistent slapping second sound cannot be taken as definitely indicative of a normal aortic valve. Vierordt assumes that in weak dilated hearts dilatation of the aortic ostium may cause "relative Klappeninsufficienz."

Cases of alleged temporary aortic regurgitant murmur are few and need very careful interpretation. My own conviction is that if such cases be followed up, the regurgitant murmur will be found permanently established at no distant date; as in Dr. Hermann Weber's very interesting case (p. 946). That dilatation of the aortic orifice does often occur is well known to every pathologist; but I have never happened to meet with such an increase in the sectional area of this orifice as to permit of regurgitation without disease of the valve; however, a few spec the kind are to be found in museums. Professor Osler (62), on authority, tells us that "the aortic orifice, which at birth is increases gradually with the growth of the heart until at one-anc it is about 60 mm. Of this size it remains until the age of forty, which date there is a gradual increase up to the age of eighty, may reach from 68 to 70 mm. Thus at the very period of life i sclerosis of the valve is most common, there is a physiological t toward the production of a state of relative insufficiency." But whe to Osler's opinion on the point before us, I find that "relative insu of the sigmoid valves, due to dilatation of the aortic ring, is a rare con he adds, "Indeed I have myself never met with a pure instance of t for in such cases I have always found the valve segments involved arterial coats." I may repeat once more that aortic insufficienc eminently a disease of old persons, but rather of persons about c middle age; there is no difficulty, of course, in collecting cases o regurgitation due to senile arterial disease,—I have such a cas my occasional observation at present,—yet the prevalent effect o disease in the old is obstruction. Again, although in elderly and in younger men the subjects of syphilis, we meet with cons and even enormous dilatation of the aorta, yet even in these case regurgitation does not generally appear unless there be disease valve itself also, for the orifice is prone rather to stiffen than t Whether then regurgitation, permanent or temporary, may arise out of a mere dilatation of the aorta, if no longer an open questi rare event; and, as the accompanying tracing shows, the incompe slight. I gather from Prof. Tigerstedt's new that in his opinion the semilunar valves are under conditions of considerable relaxation, of heart muscle or of the supporting structur Professor Sherrington (p. 466) states on exper evidence that the limbs of the valves may a other by mutual readjustments. Dr. Newt has recently investigated this matter (67).

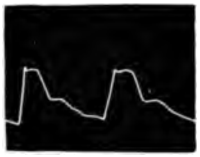
FIG. 50.—Dilatation of arch of aorta; incompetence from enlargement of orifice. Contrast with rheumatic case, Fig. 60. (Graham Steell.)

Sometimes, as Corrigan showed, on exan of the aortic valve after death from wha disease, its segments are found atrophied; tl are thin, and not infrequently "fenestrated," especially on a line to the free edge. It is alleged that those conditions are not nec morbid or mischievous; if on overlapping margins they do n rise to regurgitation. Aneurysms of the parts about the valves r discussion here, as their pathology is dealt with in the article on An Nor will I stay to discuss such pathological curiosities as morbid g polypi, and the like.

The effect of aortic disease on the other valves and orifices h carefully studied by Professor Hamilton. Aortic regurgitation observes, is "anticipated in its injurious results on the other by its own peculiar sources of mortality." From his measurements, however, the following results appear; namely, that, unless in addition to the incompetence of the valve the aortic orifice be dilated, "the effect upon the size of the other orifices is nil"; if, however, the aortic orifice be dilated, a general distension of all the other orifices is apt to follow. "Constriction of an incompetent orifice, then, exerts a salutary effect"; so far, that is, as stress on the other orifices of the organ is concerned.

Simple stenosis of the aortic orifice, in the strict sense of the word, seems to be a rare disease. I find that both Osler and Hamilton speak of it as beyond their experience. Fagge and Pye-Smith speak of it as "most rare"; Fraentzel as "ein seltener Herzfehler." As Frenchmen will not put indexes to their books, I cannot say what their experience may be. For my own part I should say with Fraentzel that the condition is rare, but not excessively so. It has happened to me to see many cases of mere aortic stenosis without any trace of regurgitation, and to have verified not a few after death, the last case being that of Professor Bradbury's, to which I have already referred. Aortic stenosis is a long disease, for life may continue under favourable circumstances until the aperture is reduced to the size of a crow quill or less. The chink by which the blood found access to the aorta in Professor Bradbury's case was only discovered on the closest search after death. It is commonly said that aortic contraction in this simple form is the result of chronic endocarditis; that aortic stenosis is connected with arterial disease which spreads down from the aorta. But in St. George's Museum are a number of cases of well-marked aortic stenosis, and in many of them the aorta seems healthy. The stenosis seems to protect the aorta in spite of the high velocity of the "choke bore." Most of these cases occur in persons of fifty years of age and upwards, in whom the incident is usually due to "atheroma." In younger or sounder persons it is often fibrotic. In these cases the effects of aortic disease upon the left ventricle are most clearly seen; it is in them that hypertrophy takes its simplest form. In so far as the aortic orifice is narrow, the inner surface of the left ventricle is protected from the pressure of "recoil."

In diseases of the aortic valve, as of other parts of the heart, our attention may be too much given to murmurs; the working calculation which we have to make is the effect of the lesion on the chambers, for by their efficiency the organ stands or falls, at any rate for a time. In stenosis the left ventricle may approach that mythical type "concentric hypertrophy." In regurgitation, especially if attended, as it is wont to be, by dilatation of the aorta, the ventricle is at least as much dilated as hypertrophied. The pathogeny of this event has been much discussed, and the outcome of the discussion is that this dilatation is due to the recoil of blood from the aorta upon the wall of the ventricle in diastole. Besides this resistance head in the arteries, that large fraction of the force of the systole which is stored up in the aorta in its diastole is expended not only upon the forwarding of the blood, but in large part also upon greater capacity, even though the height of each systole may be as before (Gaskell). The distensile force being greater, the corresponding increase follows a well-known physiological law. In like manner the increase of the muscular coat of the smaller arteries described by Sir George Johnson and Dr. Savill comes about in the course of resistance to distensile forces; and I may add that, as in the heart, this overgrowth is intimately associated with degenerative implications.

Dilatation gains on hypertrophy, as Starling clearly puts it, because, although a loaded does more work than an unloaded muscle, the amount of contraction (that is, the height of the lever) is less. The cardiac muscle may be more tense, and the contraction therefore more powerful, but it is not equal to the increased length of the muscular fibres; thus some dilatation remains, the residual blood is more, and the output less. On the next diastole the heart is overfull, but even under this increased stimulation only the normal inflow is sent out: arterial pressure is thus kept up, but work is increased and hypertrophy should follow. Not only has some of the output to be lifted again, but the backward motion of the refluent blood has to be converted into a forward motion. It is often said that the heart attains a larger bulk in aortic regurgitation than in any other disease; this is, generally speaking, true; but in chronic Bright's disease the "cor bovinum"—the "heart of a pantophile," as Voltaire called that removed from Diderot's body—may attain to no less a bulk; that is, say, 2-3 cm. at greatest thickness and 1-2 cm. at apex. It is usual to speak of this enlargement as a compensation of the defect it counteracts. There is no objection to this expression if it be remembered that it is a figurative one; all we know is that increase of function within certain limits is followed by hypertrophy; it is not in physiology only that function creates structure: yet if in respect of one factor the difficulty is postponed, the readjustment, as we shall see, brings other evils in its train. We do better, then, to get rid of these teleological connotations, and to regard the hypertrophy of the ventricle simply as the result of increased function, whatever the consequences. How does this alleged compensation break down? The late Mr. George Busk once reminded me that all muscular overgrowth may be transitory, owing perhaps to the transitoriness of all conditions less stable than the normal; he then adduced for the first time the example of the hypertrophied biceps of the file-cutter, which is said to fail after a certain number of years. But perhaps the failure is a particular instance of the general truth that a disproportionate increase of one part of a system disturbs the relations of all associated parts, and it begins to rock: hypertrophied engines in swift passenger ships mean a shorter life for the ships. Not to look beyond the immediate neighbourhood of the heart, the aorta is strained under the immoderate stress; it dilates; atheroma, the effect of strain, being usually found just above the valve, implicates in time the mouths of the coronary arteries; from the first these arteries, like the rest, are injured by the excessive percussion, and perhaps from the first the sudden and early fall of pressure

in the aorta may be greater than the higher pressure with wh
are filled on systole. Thus areas of the cardiac muscle come
blood; fatigue is cumulative, and fibroid tissue, which is more ec
to feed, supplants the active muscular fibres. If the aorta be u
to begin with, this disintegration takes place so much the soone
also the postscript to this article.]

The only other point to which I must refer in this place is t
of a persistently large residuum of blood in the left ventricle on
traction. The most recent observations seem to indicate that the
ventricles never quite empty themselves—that there is always some
blood, some difference between contraction volume and output
be so, how much more must this be the case as the work
ventricle, distended under higher pressure, increases as the cub
radius of curvature. This consideration alone, when we reco
cumulative effects of fatigue and the many incidental causes of
the heart, may go far to account for the wane of compensati
the other hand, in aortic insufficiency the ventricle contracts aga
resistance, in stenosis against high resistance, so that the output
far more in the former, as indeed the upstroke of the sphygmogra
us that it is. In the normal state the blood-pressure falls sudden
ventricle, gradually in the aorta: in regurgitation it falls sudden
aorta also; moreover, in insufficiency the pulse-rate is usually mo
That dilatation is nevertheless the feature of insufficiency rather
stenosis would indicate that mere residual blood is not the pred
factor in dilatation which is usually supposed: excessive co
volume is probably far before it in this effect. It is as card
energy begins to fail that residual blood becomes so grave a cond
dilatation. Finally, in regurgitation dilating stresses tell on the
when this muscle is relaxing, in stenosis when it is contracting.
regurgitation and stenosis occur together the results will be com
of the characters indicated for each alone, stenosis probably havi
protective effect.

The consequences of aortic disease are so often confined to
sphere that disease of this part has a character of its own; the co
pathological changes which, if the patient survive, make themse
sooner or later in other parts of the heart, need not detain us.
from uncommon in aortic regurgitation, after long persistence ev
extremer symptoms, not to detect any implication of other parts
thickening of the mitral cusps under the effects of the hypert
the left ventricle: under increasing dilatation, however, the
valve may give way, and the patient may die of mitral rather
aortic disease, although death with dropsy may occur without any
mitral regurgitation. It has been said that forcing of the mitra
with moderate regurgitation gives relief to the overstrained arterial
tion; such a temporary effect it may have for a while, but it is t
ing of one more of the gates of death.

The pathological changes in the arteries, due to their high under the heavy beating of the heart, do not differ in kind from those to be described in the chapter on "Diseases of the Arteries."

In senile aortic disease, emphysema and other evidences of decay too frequently increase the burden of the patient's latter days. The pathology of these associated degenerations is described elsewhere.

STENOSIS.—**Symptoms and signs.**—The invasion of stenosis, as of regurgitation, is often long latent. While speaking of the causation of aortic stenosis I said that this disease, standing alone, is a rare one; aortic systolic murmurs are, indeed, among the most frequent of clinical signs, but in many cases, even if organic, they signify no more than a deformation of the orifice, whether sectional area be diminished or not; again, in many cases in which this area is diminished the valve is also incompetent, and the case is no longer a simple one. To understand stenosis we must study it in its unmixed form. It has been my fortune to see not a few of these cases, and I cannot begin better than by a brief record of one of them. Mr. X., a patient of Mr. William Hall of Leeds, became aware of an increasing oppression in the chest. Mr. Hall found a direct aortic murmur and hypertrophy of the left ventricle, and was good enough to ask me to see the case. Mr. X. was a man of about fifty years of age; he had never suffered from rheumatism, his life had been anything but laborious; there was no history of syphilis nor any evidence of this infection. He had always been a man of correct and domestic habits. Nor was there any sign of kidney disease or of general arterial disease; his arterial system, apart from the aortic valve, seemed to be no older than his years. As no great improvement came about, nor was likely to come about, Sir William Gull came down to meet us, the case being even for him a rare one, and he took the greatest interest in it; so interested was he that he took occasion to call on me on a later day to talk over the symptoms again and to impress the facts on his memory. Often I recall him as he sat in my room describing with his hand an imaginary cardiographic curve in the air—the portentously long upstroke, percussion it could not be called, while the heart was heaving under the hand as the back of some imprisoned monster; the curt diastole with faint second sound; the irregularly protracted pause as if the heart were slowly gathering itself together for another effort; the deliberate rhythm, some forty in the minute, in which each reluctant beat, stout as it was, seemed as if it might be the last effort; the small hard pulse; the substernal oppression, all these features combined to make a striking clinical picture. The slowness of rate necessary to compass as much output as possible was well illustrated in a case of this kind reported by Dr. S. West, in which the pulse was 30, and in another by Dr. Parkes Weber (96) in which the heart's beats became so slow as to give rise to syncopic attacks.

For the most part aortic stenosis appears in persons beyond middle life in whom this deformity is but part of a general decay: in such

persons the compensatory reactions may be less obvious; for, to
point alone, the mass of the blood to be lifted—the cardiac ou
less in old persons than in such a subject as Mr. X.: neverth
we have seen, even the hearts of old people can attain to no in
able amount of hypertrophy; the old woman referred to on p
had a heart of 24 ounces, and apparently of good muscle.

Although it is true that the left ventricle, spared the
regurgitation, does not dilate in stenosis as it does in insu
yet it is untrue, on the other hand, to say that it does not
all; the residual blood on each contraction may be large, an
auricle gains a little in strength to meet the increased pre
the ventricle, the contraction volume of this chamber is e
and some dilatation is inevitable. The enlargement, how
more in the downward and outer than in the transverse direct
dulness does not cross the sternum, or at any rate not until t
phases of the disease. Gradual and restricted as the output may
mean arterial pressure is fairly high—the heart being usually s
systole is not only strong but absolutely, though not relative
tracted, as shown in the curve here reproduced; the pulse
"sustained." It may be that the arteries contract upon their
content. The aorta, on the other hand, is not dilated, at any rat
a direct consequence of the stenosis; if there be no arterial di
weaken it, the vessel being less distended is, theoretically at any
increased in diameter, and may be diminished. The second so
vary with the state of the valvular segments; if these be harde
sound may have the "parchment" character; but it will always
as the blood-pressure above them, even if of normal mean owin
length of systole, has not a high maximum; and unless the v
drawn nearer the sternum it will not be loud because the
area of the aorta at its orifice is diminished. It may indeed
inaudible as in J. D. (Fig. 51). The contrast between the bi
beat and the small pulse may be startling, in which respects

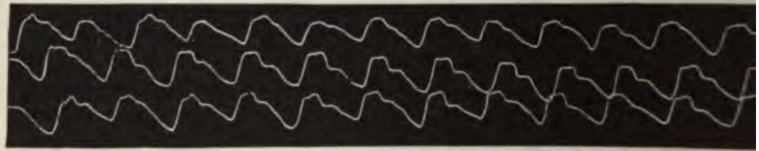

FIG. 51.—James D., æt. 46, acute rh. æt. 7. Loud syst. m. in aortic area; no diastolic second sound. P.M. No incompetence. (Graham Steell.)

differs from regurgitation, wherein the pulse, although
duration—"collapsing," has a very high maximum. In re
tion the "arterial tension" is enormous, as we see by the dama
to the structures. In stenosis, then, the protraction of the p
the cardiac revolution makes up for the smaller delivery of bl
the aorta per unit of time. In Dr. West's case, as I have said, th

rate was only 30. Sir S. Wilks again describes the pulse in stenosis as "small and slow." Yet many cases of positive stenosis with a quicker pulse are recorded, wherein the rate may be due to cardiac failure or to a call from the tissues for more blood, a call transmitted through the accelerators. In strict stenosis, then, we ordinarily have a long slow pulse with a low maximum, unaffected by raising the arm; in regurgitation, on the contrary, a short pulse not slowed, of extreme maximum pressure, and far more injurious to the arterial tree.

The murmur of stenosis may be heard widely over more than the cardiac area; it is often heard at the apex and over the aorta in the interscapular region. The character of the murmur varies to some extent both in quality and in order. Sometimes its sound vibrations are attended with others less numerous, not rapid enough to cause a sound but perceptible to touch as a *thrill*. I need scarcely say that these coarse vibrations need not indicate extreme stenosis. A thrill is often to be felt in stenosis of the mitral valve, but its position is then at or about the apex, whereas in aortic stenosis it is chiefly about the base. A thrill perceptible over a large part of the cardiac area but rather more towards the apex, is occasionally present in other labouring hearts —especially when, as in the arterio-sclerosis of old folk, the vessels become rigid while the heart itself remains vigorous and the blood-pressure high; but a thrill at the base is almost pathognomonic of aortic stenosis, whether in combination with other mischief or not. Hence we expect that the murmur also will, in part at least, be compounded of slow sound vibrations, and, whether "musical" also or not, will be noisy; we call such murmurs sawing, rough, or harsh. As the ventricle begins to give way under its toil the murmur will grow softer, possibly even to extinction; under digitalis also it may alter in quality, and the pulse may quicken in rate; then again the harshness of the murmur may return as the pulse slows down. The thrill likewise depends on the vigour of the heart; when strong, it may be felt in the vessels of the neck. As regards the order of the murmur, I have heard it sometimes in a post-systolic rhythm occupying the shorter pause; I have noted two very definite cases of this kind in private patients within the last few weeks; the first moment of systole was free from murmur, then followed a very brief murmur, and instantly thereafter a clear second sound—clear of murmur, that is, though in such cases rarely normal in tone. This is no "pulmonary" murmur.

I am interested to find that Vulpian also reports such a murmur so placed:—

> A woman, æt. 40, suffered severely from acute rheumatism; two years later she presented herself with mitral regurgitation revealed by the ordinary signs. At the base a roughish bruit was also heard; this basic bruit was placed between the two normal sounds ("entre les deux bruits normaux"). The murmur was heard also at the mid-precordial region, and upon the localisation of a rough short systolic murmur. The pulse was regular, small (rate not given) "et un peu concentré." The ascending sphygmographic line was ill-marked (très peu

accusée). Vulpian was bold enough to diagnose a contraction "sousa
"une lésion de canalisation . . . à une certaine distance audessous des
aortiques."

Vulpian does not give his reason, it may lie in the increase of
as the ventricle contracts. Constriction in this place might be
by a "presystolic" murmur as recorded by Lemoine (quoted by S
that is, by a murmur coincident with the earliest ventricular effor
is Dr. Sansom's supposition.

In most cases, when the murmur of aortic stenosis is saic
in part "presystolic," this apparent origin of the murmur is sugge
the great protraction of the "prosphygmic interval." This inter
indeed become perceptible to the finger. Ordinarily the murm
long one occupying the whole of the first phase up to diastole.

The propagation of the murmur from the second right costal c
depends much on the stage of the disease. If the murmur be l
is often loud enough to be heard at a distance from the chest—its
diffusion will be considerable, both about the basic region and
the periphery in the arteries. Thus it tends to gain an ascenden
other murmurs, and quite possibly by interference vibrations to
resolve them. When stenosis is extreme, however, it is said t
murmur may fail to reach the carotids.

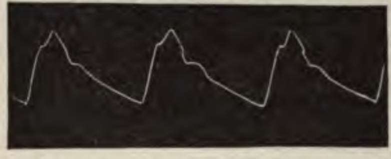

FIG. 52.—Anacrotic pulse, sloping upstroke; apex of curve formed by tidal wave; ill-marked dicrotic wave. Man, æt. 29, with rheumatic history, loud syst. m. and thrill in aortic area. (Graham Steell.)

The anacrotic and the bisferiens pulse.—It has often been sa
the anacrotic pulse (Tracings Nos. 52, 53) is so marked a pec
of aortic stenosis as to be pathognomonic of this condition.
certainly not the case; for
part, I have found the pul
crotic in so many different
cardio-arterial disease that I
not go farther than to sa
it suggests disease of the
or aortic stenosis; moreove
doubly inconstant, it is not pe
from day to day in the sam
Our attention has been draw
particularly to this matter of late by Dr. Graham Steell, who
enough to allow me to reproduce some tracings of the anacrotic
the bisferiens pulse respectively. Dr. Steell reported the behav
the pulse in four cases in which the observations on this fo
were interpreted by autopsy. The author concludes as follows:
of the four cases bore out the belief that the anacrotic puls
valuable sign of aortic stenosis, provided the physical signs corr
The fourth case taught, however, that pathognomonic value must
attributed to this pulse, inasmuch as other conditions besides
stenosis may produce it (Fig. 54). Moreover, in cases i. and iv. th
was not constant in this character; in case iii., however, unal
ness of the pulse was a striking feature of the most definite case

inasmuch as it was the least complicated. Such unalterableness of the anacrotic pulse is probably of great diagnostic value, although it may be rare."

Of the pulsus bisferiens (Fig. 55) Dr. Steell says that although cases of stenosis are so often associated with regurgitation it is not easy to find material on which to make conclusions regarding pure stenosis, yet we may assert, on Mahomed's authority, that the pulsus bisferiens is consistent with pure stenosis. In his own two cases of pulsus bisferiens there was some regurgitation also. On the other hand, in some cases of regurgitation associated with the bisferiens pulse stenosis was scarcely present, if at all (Figs. 56, 57); and again, for our yet greater uncertainty, we read that in Steell's cases the phenomenon was unequal on the two sides, and in one of them chiefly unilateral, the other radial assuming the character very occasionally

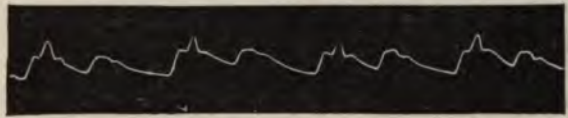

FIG. 53.—Florence ——, æt. 28, rheum. Loud syst. and diastolic m. in aortic area. Death from cerebral embolism. Anacrotic pulse with bigeminal or alternating rhythm, possibly due to digitalis, although none taken for two days. P.M. (Graham Steell.)

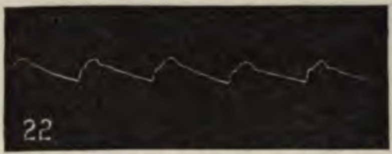

FIG. 54.—Anacrotic pulse-tracing from case of mitral stenosis without aortic stenosis. P.M. (Graham Steell.)

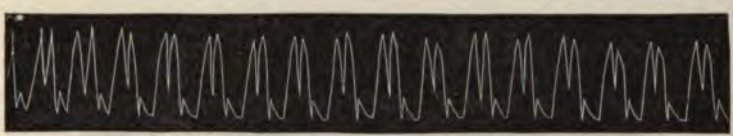

FIG. 55.—Margaret G., æt. 25; rh. æt. 14. Exemplary P. bisferiens. R. radial, double beat plainly felt; L. radial, ordinary tracing of aortic incompetence. Loud syst. m. and thrill in aortic area; diastolic m. P.M. Stenosis of aortic orifice with incompetence of valves; no explanation of difference between radial pulses. (Graham Steell.)

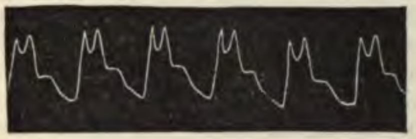

FIG. 56.—Aortic incompetence without stenosis. Man, æt. 29, with history of rh. at æt. 20. P. bisferiens. P.M. (Graham Steell.)

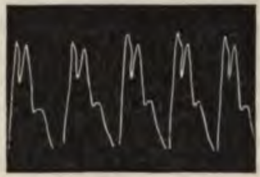

FIG. 57.—Bisferiens pulse in case of aortic incompetence without stenosis. P.M. (Graham Steell.)

and imperfectly; a careful examination of the arteries concerned a
explanation of this peculiarity (Figs. 58, 59). In two cases, mor
phenomenon was manifested in the one on the right side, in the
the left. Dr. Steell concludes thus :—"We are unable to explain
of production of these pulses; and I do not think we are war
affirming either that the anacrotic or the bisferiens pulse is the dir
of aortic stenosis; both pulses are found, however, so often in a
with aortic stenosis that we cannot deny them diagnostic value
two the anacrotic pulse probably possesses the greater d
value."

As regards the anacrotic pulse Dr. Sansom virtually comes to
conclusion: he emphasises the deduction that a persistently anacr
means organic disease, whether aortic or chronic renal; and tha

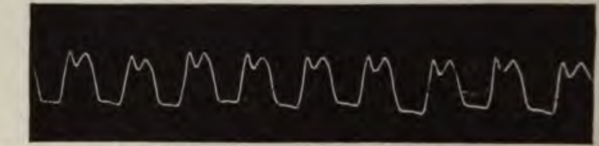

Fig. 58.—Left radial. C. E. W. æt. 18. Loud syst. m. with thrill in aortic area; aortic d
fingers clubbed; patient stunted in growth; no rh.; no chorea; probably congenital
supervening septic endocarditis. (Graham Steell.)

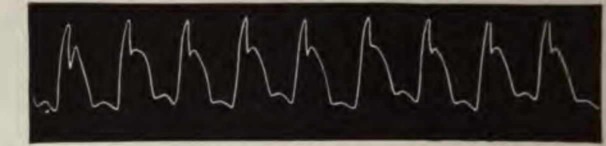

Fig. 59.—C. E. W. Right radial, showing the limitation of bisferiens pulse to one radia
(Graham Steell.)

of doubt an anacrotic pulse might signify that a systolic murmu
base of the heart is not anæmic. In aortic stenosis, as it seems
anacrotism is easy to explain: the pressure in the aorta is low
normal, that within the ventricle is much higher; at the first
then, of the opening of the valve the blood issues readily, bu
stenosis throttles the wave the increased velocity of the blood is
acted by the rising pressure in the aorta, and the farther delivery
slower and more laborious; though so long as the heart is str
pulse is regular. Perhaps the most general expression under whic
bring the anacrotic pulse is that during systole the flow from the
the periphery is at a slower rate than that from the ventricle
aorta: in aortic stenosis the current issuing from the choke bore
high velocity; that from the aorta to the periphery, however, is sl
as the blood occupies a relatively large channel, an adverse c
which may be exaggerated by increased peripheral resistance; th
the other hand it is diminished by arterial rigidity, in which case, a
the wave is accelerated, the blood-current is not.

Pain is not a constant feature of aortic stenosis; as in regurgitation it probably depends on aortitis, and is much aggravated by cardiac stress. In my experience neither anginal pain nor the sense of substernal oppression are so great, if as frequent, as in regurgitation. Still it is often pain, either acute or oppressive, which sends the patient at first to the physician. It is rarely severe, but may run down the left arm and serve as a warning to the patient when he transgresses the limits within which he must conduct his life. In some cases, however, the angina, whether in obstruction or regurgitation, may harass the patient even when at rest in bed (Douglas Powell, vol. vi.). Here I may digress so far as to state my opinion that the seat of the distress in angina pectoris is in the aorta, and not in the heart.

Dyspnœa.—In aortic disease dyspnœa is not so frequent nor so prominent a feature as in mitral disease; and this for obvious reasons. The substernal oppression of stenosis and of regurgitation is often falsely called dyspnœa; it is rather the complaint of the aorta and of an overworked left ventricle in distress.

There is not the same eccentricity of symptoms in stenosis that there is in regurgitation; there is less tendency to gastric perturbations, to headaches, to pangs having the degrees of angina, to cough (cough depends rather on a dilated aorta), or to faltering of the mind and memory.

Diagnosis.—Latent as stenosis of the aortic orifice in its early phases may be, on the other hand I need not repeat that to infer this deformity in every case of basic systolic murmur would lead us into error. Not even in persons of advanced years, in whom in all probability such a murmur does indeed signify disease of this area, is it to be assumed, as too often it is, that the aortic orifice is positively contracted. We have already seen that aortic contraction without regurgitation is a rare condition; yet systolic murmurs in the aortic area are of the commonest of clinical events: in young persons they are usually due to perversions of the blood, in the elderly to atheromatous disease. We have then in the first place to decide whether in a given case an "aortic systolic murmur" is of the kind known as hæmic, or due to atheroma or other chronic arterial disease, such as the syphilitic. Of murmurs due to the acuter degrees of endocarditis I do not speak; they are discussed in the chapter on "Acute Endocarditis" (p. 869).

In the first place, then, is a given aortic systolic murmur, hæmic or organic, dynamical or statical? Neither age nor sex is conclusive: a young woman may suffer from aortic stenosis of a fixed organic kind, without regurgitation, and without any history of rheumatism, chorea, or other constitutional disease; how are we to decide that in her the murmur is one of stenosis of the aortic orifice? Well, in the first place, there may be no definite signs of anæmia, no venous hums, no characteristic blood changes, no change of intensity on varying her position; the first cardiac sound is inaudible. In anæmia a murmur may be loud, occasionally even harsh, and in stenosis the murmur may be soft; but a sawing sound, especially if associated with a thrill, would strongly suggest organic disease.

In stenosis the apex of the heart is perhaps a little displace vertical direction, and the cardiac impulse not merely forcible, no violent, but steady, long, and heaving. The over-action of ventricle may be manifest, yet the cardiac dulness scarcely increas versely. A substernal oppression may make itself felt on exe even during rest, which differs altogether from the painless a panting dyspnœa of anæmia. This oppression often amounts and may then run into their left arm. For a broader discussio part of the diagnosis the reader is referred to the article on "Chlo 503. Again, although Bright's disease be not present, nor general disease, the pulse may be anacrotic; this feature of the pulse long plateau would set aside that extremely rare affection pu stenosis. Again we shall endeavour to ascertain from the hi the case whether the disease is congenital. Can such a murmur of mitral regurgitation, or even of mitral contraction? As reg latter, the propagation into the carotids, perhaps down the aorta and the position and time of a thrill, are important distinctio if we suppose a mitral disease insufficient to cause symptoms of retardation; the murmur, again, may be comparatively feeble axillary line, and is usually so at the apex. But why stenosis? not be that a spur of a diseased valve in the blood-current is th of a murmur within an orifice of at least normal dimensions? the cardiac hypertrophy be accounted for by arterial resistance, in the aorta or elsewhere; or again may there not be an aneurys of the sinuses of Valsalva? This last chance cannot be eliminat against it is the hypertrophy of the left ventricle, which is not a of these aneurysms; and (hereafter under regurgitation) I shall hav something about retardation of the radial pulse in aneurysms whi have some importance in this respect. Moreover, a harsh murmu in our general experience of aneurysms, characteristic of them. ever, the systolic murmur be less harsh, this point loses its fo exclusion of aneurysm rests only on the rather uncertain ba moderate ventricular hypertrophy, the absence of retardation small volume of the radial pulse. Again, may the murmur be ge in the aorta without stenosis? I have watched so many cases aortic dilatation to their close in death in which neither a systolic nor any other murmur ever appeared, that I hesitate to say th murs arise in the absence of implication of the orifice. That should form as the blood spreads into the larger channel seems and that they should thus set up murmurs seems also likely; ye cases in which I have followed organic systolic aortic murmurs to t mortem table the orifice has presented disease amply sufficient caused the murmur. Aortic systolic murmur with valve and virtually normal is outside my experience; dilated aorta, even in degrees, without murmur is abundant within it. Finally, is the actually constricted, or is it merely deformed without constrictio stenosis I think that there are three tests of the condition: the of hypertrophy of the ventricle, the volume of the radial pulse, and the diameter of the aorta.

Rosenstein, following no less an authority than that of Traube, asserts that in aortic stenosis the ventricular impulse is weak or even imperceptible; on the whole this opinion is contrary not only to my own experience, but to that of others; moreover, we must admit that it may not be possible always to distinguish between cases in which a murmur is generated at the orifice without constriction in the positive sense and those of stenosis proper. If there be any power of response in the heart at all it seems inconceivable that an increase of resistance such as we contemplate should fail to produce hypertrophy, and that it does so is a matter of certain observation; moreover, in the cases in which signs of hypertrophy are absent the pulse is of normal or excessive rapidity. If an organic systolic murmur be heard at the aortic orifice, if the pulse is 70 or over, and there is no hypertrophy of the left ventricle, I should say that the disease of or about the orifice has not the effect of stenosis, or, if it has, that the nutrition of the heart is failing.

Prognosis.—It is said that the forecast of aortic stenosis is of all heart diseases the least unfavourable. No doubt this is true if we bulk together all organic murmurs heard at the aortic orifice; but this is a pell-mell classification. We see, it is true, well-to-do old ladies leading tranquil lives up to fourscore years or more with systolic aortic murmurs of a quarter of a century's standing; as we see such persons with arteries reduced to trees of coral, yet living the length of the human span. I suppose that such survivors persist in virtue of fair cardiac hypertrophy, and of the absence of aneurysms on their cerebral arteries. In my eye as I write is an old lady whose carotids were jumping to the eye, and whose radials were as tobacco-pipes fifteen years ago, who still pursues the unbroken tenor of her existence with no more to trouble her than a slight dry gangrene of the toes which left her lame half-a-dozen years ago. In these patients, however, the demands of life are of the narrowest and the lightest; the expenditure is almost nothing. In Professor Bradbury's case of stenosis—that in which the issue of the blood was by an almost imperceptible channel — the heart of 24 oz. was evidently able still to drive an attenuated stream of blood through this tiny hole at a velocity, perhaps, of some four metres per second; so that the blood column in the aorta was sustained for a long time at a pressure compatible with life. When we regard such cases, and those again in which the aortic mischief sets up corrugation rather than strict stenosis of the orifice, the prognosis seems better than in that next best disease, mitral regurgitation. But a broad division may be made which will show aortic stenosis in a less favourable light. If we take patients under fifty-five years of age we shall find the prognosis much worse. In most of these cases the stenosis is stenosis proper and of a kind to lead to further constriction; it probably consists in fibrous inflammation about the ring and the limbs of the valve of a progressive kind. In them the prognosis would seem to be rather worse than better for the absence of atheroma: moreove young the system is more exacting in its demands, and the is not becalmed in the senile torpor of body and mind. Much of course, on the rate of a subinflammatory process, but my sion is that a person who in young or middle life begins to suffe from the symptoms of aortic stenosis has but a few years to live. duration of its latent period it is of course hard to judge; the may be detected by chance, but such discoveries are too irre provide materials for prognosis. Old folks apart, then, my esti the duration of a case of stenosis is not a sanguine one. T phase may be by dilatation and backward pressure; but the usu of death is exhaustion of the left ventricle and syncope, or dege of it and asystole: the former being the predominant and somet only condition, as in the case of a boy (mentioned above by D. Powell) who succumbed and died suddenly while running. necropsy extreme aortic stenosis was discovered for the first time

The treatment of aortic stenosis is considered at the end article.

REGURGITATION.—**Symptoms and signs.**—Like stenosis regu is often covert in its invasion; moreover, the cases are many in w signs and symptoms of this disease are found without apparent c have said that aortic regurgitation is not the ordinary course of e elderly and atheromatous persons; a systolic murmur in this are ordinary result of atheroma. Regurgitation is practically alway panied by a systolic murmur, but I repeat that such systolic m do not always or even usually indicate positive stenosis; as indeed readily infer from the volume of the pulse.

Whether the mischief be due to past rheumatism (acute end is dealt with in another chapter), to strain, to syphilis, or to at the symptoms and signs are much alike. But in the two latter expect to find, and we generally do find, by other signs and sy that the cardiac disease, pressing as it may be, is but a part of spread arterial disease [*vide* "Diseases of Arteries," vol. vi.]. over, in the atheromatous and syphilitic cases incidental disasters, apoplexy or embolism, are more likely to befall the sufferer.

Pulse.—Aortic regurgitation is sometimes revealed, either patient himself, or to an observant friend, by the characters pulse, a very prominent and peculiar feature of the malad it usually betrays itself in the first place by substernal oppre symptom which we have already considered under stenosis.

The incomprehensible statement is commonly repeated that i regurgitation the arterial tension is low, and this in face of evid tensile strain witnessed in like degree in no other disease. This strain is due to the stress of the hypertrophied left ventricle u arteries, a stress often not mitigated by the bar of stenosis or by tection of tone: the arteries are large and slack. Under condi high pressure, as in Bright's disease, if the arterial charge vary within narrow limits, high as the mean pressure may be, the maxima and minima are not widely apart. Under these circumstances, especially if the muscular coat of the arteries be hypertrophied, tone secures something like a uniform adaptation of the vessels to the blood. If tone be deficient in an arterial system otherwise normal, we find a wide divarication of the maxima and minima; but this is temporary and harmless. It is otherwise when, as in aortic regurgitation, the condition is both exaggerated and persistent. In this case tensile strain, acting both in the longitudinal and transverse directions, widens and lengthens the vessels, tends to split them across or along; and arterial tone, weakened by strain or anæmia, or put in abeyance by some reflex mechanism, is unable in any conservative degree to adapt the continent to the content; the extremes of volume are too far asunder. The circulation changes into the form of a series of discontinuous discharges, as if from a catapult. The well-known tracing of the radial pulse in regurgitation shows a high and violent percussion, usually with an inertia "crochet" at its summit, and as sudden a descent without plateau (Fig. 60). Now it is not necessary to make a long series of observations to ascertain whether in a number of cases the mean pressure is higher under these extremes than in an equal number of cases of hypertrophy of the left ventricle without regurgitation; nature has given us the information in the state of the arterial tree, in the lengthened and dilated vessels; strains which eventuate in general arterial disease, especially in the parts most exposed to the intermittent pulses of the blood. These results justify us in supposing that in aortic regurgitation the mean arterial tension is higher than in any other disease; though as, for some obscure reason, it seems to be less modified by tone, a deduction may have to be made in this respect. The effect of elongation of the arteries is to throw them into curves; and as these are straightened at each diastole, the vessel is then thrown out of its bed with a visible and palpable jerk. The wife of such a patient told Sir Thomas Watson that for some time, on taking her husband's arm, she had felt this uncomfortable jarring.

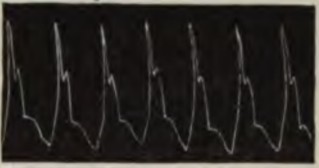

FIG. 60.—Margaret E., æt. 38; rh. æt. 17. Typical pulse of aortic incompetence, showing exaggerated percussion wave, deficient dicrotic wave, and unsustained tidal wave. (Graham Steell.)

Whether in a normal peripheral artery, such as the radial, the pulse should be visible is a matter of doubt. In some thin people, in whom a fine skin allows the radial artery to be seen, the pulse is perceptible to the eye, especially if its tone be slack. It is alleged that this beat is made visible by the tension of the skin over the vessel, and that were such a vessel without dimple or dint the pulse would not be visible. In arteries such as the temporal, which are without much cushion, elongation takes place more readily; and in men still young the temporal is often thus thrown into curves which reveal the pulses clearly enough, though in all other respects the vascular system may be free fr hint of disease. After the tension of aortic regurgitation has b tinued for a longer or shorter time, all the arteries exhibit the jar pulse of which I have already spoken—"danse des artères," as the call it; they start out of their beds with each pulsation. "Someti whole of the patient's body," says Watson, "nay, his very bed, is by the strong shock of the heart during its systole." In many ca jerking is well seen in the tonsils. On raising a limb—the arm, for i —to a vertical position, the refluent character of the pulse beco more apparent, for obvious reasons; and if in any case of the kind t chief or the incidents of it be not such as to produce this characte horizontal limb it will surely appear in the limb when raised. On r limb the pressure in the peripheral vessels may fall to such an ext the pulse may actually disappear; though entire disappearance is always due to some constriction of the vessel at a higher poin the flexure of a joint or by the fold of a garment: raise the ar patient while he has an overcoat on and the radial pulse may remove the dress and the pulse may persist. Dicrotism does appear so regularly as one might expect; in the tracing, if not finger, it is often perceptible. We might have expected that w loss of the support of the aortic valve this recoil would wane appear; accordingly in the degree of its persistence a prognos has often been sought, but sought in vain: we find a dicroti sometimes in the least promising cases; the subject needs further gation. [For other sphygmographic tracings the reader is referre paragraphs on the bisferiens pulse, etc., pp. 930-932.]

It is the function of a healthy heart and healthy vessels to at each beat the maximum of blood displacement with the m alteration of pressures. At each beat the heart leaves a portio energy in the arterial tree which, given out again between the pul converts or tends to convert the intermittent pulses into a con flow: it is plain that in aortic regurgitation we have the very c —the maximum of pressure disturbance with the minimum of translation. Thus, in a well-marked case, in no part of the arter is the flow made continuous, not even in the capillaries; and Q "capillary pulse," although not peculiar to aortic regurgitation, characteristic of it. If in any malady, even in health, the tone be so low that storage of cardiomotive force in the elasti is defective, the capillary pulse may be seen. One of my once demonstrated to me the capillary pulse in his own perso in health; he told me that it was habitual in him. Dr. Waller may be detected in many normal persons, but that in its extremer it is characteristic of aortic regurgitation. On the other hand, i cases of this disease the capillary pulse is not to be seen. T presence or absence is of prognostic value is not yet known; w not hitherto connected its phases with the course of the misch do we clearly know why tone in this disease is so low. The readi of obtaining the reaction at the bedside is to press highly upon one of the patient's finger-nails with the point of a pencil, or to depress its edge with one's own nail; with a little management the pulsation becomes visible. It may be visible also in the vessels of the retina, or in the areola about such an eruption as urticaria. The same evidence may be obtained again by pressing a glass slide upon the mucous lining of the everted lower lip; or the skin on the forehead, or elsewhere, may be rubbed until the cutaneous vessels dilate, when their visible pulsing will prove how great is the factor of vascular tone in integrating the circulation. In aortic regurgitation tone yields to tension, or in the peripheral vessels atony may be due to lack of due nutrition.

Retardation of the pulse.—It has been currently reported that in aortic regurgitation the arterial pulse is retarded. Fagge held this opinion and many other physicians likewise, among whom is an observer so distinguished as Sir William Broadbent. As this proposition has always seemed to me to be contrary to observation, both physiological and pathological, it must receive some closer attention.

In the normal heart there is an interval of about 0·1 of a second between the beginning of the ventricular contraction and the carotid diastole. This interval (the Anspannungszeit or prosphygmic interval) has been carefully studied by Chauveau and Marey, Hürthle, von Frey, Keyt, Tigerstedt, Chapman, and others; and its interpretation is that in this interval the intra-ventricular pressure is rising to that in the aorta; that not until the contraction pressure of the left ventricle equals the resistance of the column of blood in the aorta does the aortic valve open and the aortic diastole occur. Generally speaking, in the normal course of ventricular systole the relative pressures in ventricle and aorta preserve the same proportions, and the prosphygmic interval is invariable; but in aortic regurgitation, the support of the valve being removed, the pressure within the aorta is not sustained, and the relation of the pressures is altered: virtually the aorta and the left ventricle become one chamber. Where there are no differential pressures there can be no differential times, and the diastole of the aorta must be coincident with the first contractile effort of the cardiac muscle. On the other hand, the moment the heart relaxes the pressure in the aorta falls to that in the ventricle. How then can there be retardation of the radial pulse? How can the radial pulse be delayed beyond the time of the velocity of the blood-wave? Lest I should be wrong somewhere in these suppositions, I have referred the matter to Dr. Chapman of Hereford. Dr. Chapman, in supporting the arguments used above, points out that Keyt independently foresaw and demonstrated this order of the phenomena in 1879. In 1887 he repeated the same conclusion, that "immediately upon the contraction of the ventricle the blood-pressure in the aorta begins to rise." Dr. Chapman renders me the service of tracing the history of the alleged retardation in aortic insufficiency. Dr. Henderson, he says, first started the notion in 1832. He was followed by authors of no less ascendency than Flint and Walshe. Keyt explains the discordance in this way:—"The enlarged ventricle suddenly filling from both the aorta by reverse and th by direct flow, communicates a shock so marked as to be for systole. This impulse occurring in the first part of diast preceding the arterial pulse at such a distance, gives the in of enormous delay of the pulse." In his Goulstonian Le find that Dr. Chapman dealt with the whole question, and gav over, in one such case, the actual measurements, when the between the systole and the radial pulse was 0·039″ to 0·031″ of the normal of 0·080″. Now if I am asked why I tre long upon the reader's time and attention with details so as these, I reply that in nature there is no large and small; for some few years I have anticipated that by such measuren these a diagnosis might possibly be made between simple aortic re tion and aneurysm of a sinus of Valsalva: unfortunately no t has yet come under my notice. Perhaps ere long some skilled c entering into this controversy, may light on a case of aortic re tion in which the radial pulse is considerably retarded; and f it up to the post-mortem table, may decide the presence or absence aneurysm therewith. Again, in double aortic murmur, in murmu and regurgitant, can we tell by these means whether the direct mu due to stenosis proper or merely to a broken blood-stream large o Without returning to what I have said under the head of stenosi guess that, whereas in stenosis proper without regurgitation the of the radial wave is retarded by conditions the opposite of regurgitation, a persistence of the normal interval between cardia and full radial diastole may indicate a combination of sten insufficiency, the pressure in the aorta which the valve is no lon to sustain being kept up more or less by stenosis of the orifice reader is here referred to the postscript.]

The characters of the pulse are well known; the gifted phy whom we owe most of our knowledge of this subject has given a me description of them. Corrigan compared the pulse of aortic regu to the "water hammer," a toy in which water, imprisoned in an e tube, falls from end to end, on every turn of the tube, with With some such thud the charge of blood is shot along the How this effect is intensified by raising the limb, and the effect i the vessels themselves, we have seen already; I have still to some other characters which are not without interest.

In cases of extensive arterial sclerosis, or at any rate of scle the radial and brachial arteries, the stiff walls of the vessel do not with the sudden ebb of the pulse wave as a comparatively norma does. Nor, indeed, can the arterial diastole be so well marked unless stenosis be present, the stiffened arteries will vibrate or the jarring in the carotids and contorted brachials will be plain e

The pulse during the more stable phases of aortic insu is regular. This is the rule, and a very important rul Trivial as an intermittence of the pulse may be in a heart, in the disease before us it is of grave significance; as grave as it is in pneumonia or in enterica. An occasional intermittence may be of no ill omen; but recurrent slips unmistakably indicate dilapidation of the heart. An irresolute or dropped beat is a far more serious event in aortic regurgitation than in stenosis, or in mitral disease: in the first case, as in "fatty heart," it is more likely to indicate a failing than a merely faltering heart, or a passing inequality in blood delivery. As in other disorders, the heart may fully intermit or contract so feebly that the pulse either fails to reach the wrist, or is but a flicker there. Irregularity of the pulse is a warning of like omen. In the aortic insufficiency of cardio-arterial disease, intermittence occurs earlier than in that of disease more strictly cardiac, at least such is my experience.

The sudden distension of the collapsed and inanimate arteries gives rise to signs which are perhaps something more than curiosities; the chief of these is the sign of Duroziez. We remember that for the most part murmurs are produced in the arterial system by the passage of the blood into a wider channel, when fluid veins are generated. If then pressure be made on an artery in health, say with an edge of the stethoscope, these conditions are fulfilled and a murmur is set up. This phenomenon is intensified in aortic regurgitation, because in this state the walls of the arteries being slack vibrate more readily, as may be conveniently observed on the femoral artery. But in aortic insufficiency, as Duroziez pointed out, there is something more than this: the artery gives out not only this single murmur, a murmur of its diastole, but a murmur on its systole also; there is a double murmur, and this double murmur cannot be obtained in the normal state. As French writers have a confusing habit of taking the word "bruit" to mean either tone or murmur, it is well to say that a tone is produced by the diastole of a normal artery near the heart, such as the carotid—a tone to be heard on light pressure of the stethoscope; but here we are discussing not the tone, but a murmur artificially produced by stronger pressure; and in aortic regurgitation this murmur is followed by a second murmur generated on the arterial systole or collapse. The causes of this latter murmur are unknown; it is easy to show that it is not dicrotic, but whether it be a "recoil" murmur as surmised by François Franck, we cannot decide. To get it clearly, Potain directs us to press on the artery with that edge of the stethoscope which is farthest from the heart, so that the whole wave, if it be a recoil, passes under the base of the instrument. He adds that for its production there is a "most favourable point of pressure," a degree between too light a pressure and obliteration of the vessel, which is, of course, to be discovered in each case at the moment of examination. Now it is said that the second murmur—Duroziez's murmur—dies out as compensation fails; if so, it is not a mere curiosity: in any case attention to such incidents as these encourage that painful research in clinics which is the only way to the increase of knowledge. Duroziez's phenomenon is not, I think, peculiar to aortic insufficiency, though

Vierordt says it is; I think I have found it under other atonic c when the arteries are unduly vibratile: if so, all it tells us is diastole is brusque and the systole "collapsing."

If again the stethoscope be lightly laid on an artery of th the carotid, a "tone" may be heard on its diastole, and not infr on its systole likewise—"the double tone"; this, though char of aortic insufficiency, is certainly not peculiar to it. If pressure a murmur occurs, as we have seen; or if an aortic direct murmu sent, the tone is replaced by murmur without the use of pressure. under normal conditions, on systole of the carotid the second the heart is audible; now, in aortic insufficiency, and in sten second sound is usually lost. In the smaller arteries, under nor ditions, the tone of their diastole is inaudible; but in aortic insu an arterial diastolic tone may often be heard down to the sma distant arteries—in the dorsalis pedis, for instance; so that I h in the habit of guessing the amount of the regurgitation from tensity of this tone in the femoral artery. Arterio-sclerosis, tends to reduce it. The same phenomenon occurs in anæmia, fe other states in which the artery is slack and the diastole sudde murmur of aortic insufficiency is often heard in the carotid, bu means always; the conditions of its propagation thither are clinical moment, in so far as they may help in the discrimin aortic from other diastolic murmurs.

The Heart.—Of the dilatation and hypertrophy and their signs has been said already in this and other chapters that I will not dw the subject. I may repeat that the enlargement may be greater insufficiency than in any other disease; and usually, at least, is unmi Perhaps its size is only rivalled in certain cases of chronic Bright's In young persons with soft ribs the cardiac area may become pr In cases of doubt it is better to lay the ear direct upon the the chest, whereby the heaving impulse is more readily apprecia is, I suppose, conceivable that in slight insufficiency hypertrop coexist with but a nominal degree of dilatation. Dr. Sansom us that the less the element of dilatation the more "tria is the apex area; the superficial area of dulness is extended wards and outwards, and does not extend far in a transverse d Although a dull area corresponding to dilatation of the aorta may the region of the manubrium sterni, and may transgress it to t the ventricular dulness may not be enlarged to the right; or no rate until in some grievously protracted case the chambers of t are involved in a common defeat. François Franck points out t apex itself may be "dicrotic"; the first shock being due to the the second to the propulsion of the blood (*vide* p. 940). Sir W. Br gives us the useful warning not to mistake the systolic recession costal spaces, due to atmospheric pressure acting upon the sp by the diminution of volume of a large heart, for a sign of adher cardium. In cases of arterial disease the observer will not for causes of hypertrophy may have been in operation for an indefinite time before the establishment of regurgitation. In such cases the hypertrophy cannot be taken as a direct measure of the insufficiency, which accident may be recent and inconsiderable, the chief part of the changes being attributable to the common causes of both; however, whether recent or of long standing, cardiac enlargement, considerable as it may be, is not so large as in young and sounder persons. It is in the aortic insufficiency of the young, due almost always to rheumatism, that the huge hearts are found which lift life along for many years.

Sounds of the heart.—In a very large proportion of cases of aortic regurgitation the first sound is impure if not actually replaced by a murmur; whether there be positive stenosis or not, insufficiency is generally accompanied by such changes in the structure of the ostium as to give rise to a direct murmur also. In arterial disease the occurrence of a diastolic without a systolic murmur is very rare, as the regurgitation arises incidentally in the course of the atheroma. In strain or rheumatic injury the murmur of regurgitation may exist alone, at any rate for a time; yet even in these cases the first sound is seldom pure. At the apex the first sound is usually prolonged, especially if there be coincident stenosis; and it takes a more "booming" quality as the hypertrophy increases. The direct murmur is usually but not always carried well up into the carotids, so that the carotid diastolic tone is replaced by a murmur, or even by a thrill; yet sometimes neither sound nor murmur is heard with their systole. The second sound at the pulmonary cartilage is unchanged, unless the whole heart be thrust still nearer to the wall of the chest, when it will seem accentuated. The second aortic sound in aortic regurgitation has not been studied very precisely. If it persist with a regurgitant murmur, it is said, a little too readily, to be the sound of the pulmonary valves only. That this is not the case seems to be proved by its frequent propagation into the carotids. Again, it is argued that if the aortic second sound coexist with a regurgitant murmur there is still a substantial area of valve closure, either by fractional parts of the valve or by the establishment of a measure of stenosis which in aortic insufficiency may be conservative. On the other hand, though the disappearance of it may be of ill omen, it is certainly incorrect to say that persistence of the second sound always means a moderate degree of regurgitation. These are points which need verification, but after all sounds and murmurs make but a part of diagnosis; in quickly beating hearts indeed such points are inappreciable. I am disposed to regard persistence of the second sound, in some cases at any rate, as due to the sudden systole of a slack and vibratile aorta, such a tone as that of the systole of the femoral in this disease. It may be audible in cases where the valve is quite disorganised.

Murmur of regurgitation.—If, on the one hand, it be true that this murmur is usually very definite in its characters and its meaning inevitable, it is none the less true that, with the exception of mitral stenosis, it is the murmur most frequently overlooked; and not by pupils only, but by experienced practitioners. Sometimes the lurks in unexpected places; sometimes its quality is so soft ar cent that a quick ear is required for its detection, especially if systolic murmur precede it. If it be both soft and aberra site even a skilful observer may be deceived, at any rate at fi only may the murmur of regurgitation be soft and distant, and in strange places, but it may be aberrant in time also. It ma fractional parts of the diastolic period, and not always the in Like the murmur of mitral stenosis, it may be perceptible only in t or final third of the long pause; and if discovered accidental rhythm, before the advance of secondary changes, it might deceive elect. One such case I remember which divided three hospital p in opinion. One inferred mitral stenosis; two held to aortic r tion. Whether either opinion was afterwards verified I cannot s Douglas Stanley described an interesting case of the kind at a bra ing of the British Medical Association. In this case a diastolic mur immediately on diastole, a murmur of aortic regurgitation well at the base; at the apex was heard a mid-diastolic murmur, than that of the base, and not heard outside the mitral area, whi before the first sound. After death the aortic valve was seen but two cusps, and these involved in a mass of vegetation. T valve was healthy. Both murmurs were clearly aortic.

Loudness of murmur is no indication of severity of le reverse is rather to be anticipated. A loud murmur generally s vigorous heart; and a refluent stream returning at a high velc set up more active veins in the ventricular content than a larg falling back through a large opening at a low velocity. If a previously loud fall in intensity we may be apprehensive Sudden ruptures of the valve often give rise to very loud audible over a large area of the chest; in such cases the murmur audible to bystanders, and even to the patient himself. In the of stenosis a regurgitant murmur is louder, other things being e the velocity of the refluent current is greater. In these cases a fairly sustained pulse is associated with a sawing double murmu returning through a perforation of a limb of the valve is said to be with a piping or mewing sound; or it has been compared to the of chickens. A murmur direct or regurgitant, audible withou with the wall of the chest, is always aortic—an inference some diagnostic value.

Prof. Sewall of Denver has investigated the behaviour of al murmurs under increasing pressures of the stethoscope. He s murmurs of aortic stenosis audible at the apex disappear under there, and are herein distinct from mitral regurgitation. Also th dilatation of the aorta or aneurysm apart, the murmurs of aortic tation may be annulled by pressure at the base but not at t "Inorganic murmurs" at the base, he says, can all be ob by pressure; and by the same means used in the second rig space close to the sternum the normal second sound can be stopped, unless the aorta be so dilated as to be in contact with the wall of the chest.

The student is often directed to track a murmur to its origin by shifting his stethoscope along the surface of the chest from one area to another, in order to note where one murmur dies and another is born. This is a misleading device, only to be used by skilled observers. A murmur, like a river, may run underground in part of its course, the conditions of conduction differ from place to place, and one and the same murmur, as the stethoscope travels, may so wax and wane, as the structures about it vary in conductive capacity, as to appear twofold. Many a misapprehension thus arises as the observer slips the instrument diagonally upwards; a murmur heard at the apex disappears to reappear at the aortic cartilage; and thus a murmur generated at the aortic orifice only may be regarded as indicative of two lesions. Another error is to assume that the murmur certainly follows the direction of the blood-current; the blood does not run in the air as water over gravel; the murmur we hear is due to the vibrations of surrounding structures—chiefly the walls of the heart—set up by the vortices within them; the heart is the fiddle, the blood is but the bow. We must rid our minds of these conceptions of blood running here and there in the chambers, as if it were from a water-cock into a pipkin, and realise that the walls are thrown into vibration by molecular collisions in a plenum.

Of aortic insufficiency with regurgitation, but without a murmur, I know nothing; but murmurs which can be extremely soft may in very rare instances be evanescent. Weismayer, in a paper which has been much quoted, accepts such statements, a little uncritically I think, and proceeds to explain them. Dr. Hermann Weber's case (*vide infra*) is an example of the manner in which in incipient cases the murmur of aortic insufficiency may cease for a time with the insufficiency which gave rise to it; again, like any other murmur, that of insufficiency may wane with the heart in which it is generated; but that with a persistent insufficiency regurgitant murmurs come and go in a comparatively vigorous heart is contrary to experience, or at any rate to mine. As testing exceptions I may refer to a case reported by Dr. Saundby, and to another reported by Dr. Musser. In Dr. Musser's curious case the corpora Arantii had been transformed into calcareous buttons (4 mm. by 2 mm.). During the formation of these excrescences regurgitation took place and a murmur was generated; but as they wore down and the free surfaces became faceted, after the manner of gall-stones, the incompetent valve became again competent.

Whether regurgitation is prone to occur in dilatation of the aorta with an unimpaired valve, and without aneurysm of a sinus, has been considered already (p. 922). Relative insufficiency at the mitral orifice is well understood and by no means rare; but this is known to depend upon the conditions of the muscular and tendinous attachments of the

limbs of this valve, a kind of attachment which does not ex
aortic valve.

With this problem is bound up that of "intermittent aorti
tation" considered above; for these cases are said to depend al
ditions of the aorta rather than of the valve. I believe that i
intermittent aortic regurgitation the valve is nearly always
but that in the earlier stages, say in disease of one of its
valve becomes able, by mutual accommodation of its parts
to close the orifice until the blood-pressure becomes excessive
other physical change supervenes. Such a patient may inde
amined at a time when the valvular disease is latent, or is not
by a murmur at any rate; and in such a case a grave error of
might be committed. The following case, resting on the au
Dr. Hermann Weber, is most instructive :—

A very active young man, æt. 32, of weak muscular develop
examined by Dr. Weber on arriving at a height of 8000 feet.
aortic sound was replaced by a musical murmur at mid-sternum and
the right. The first sound was rather indistinct. The pulse was
feeble, but not characteristic of aortic regurgitation. On the followi
murmur had disappeared; the heart sounds were normal, and the
Two days later, at 9000 feet, the same murmur became audible; a
manner disappeared on the day following. Further climbing was
and he returned to work in good health. Seven years later the p
of "Herzschlag."

Regurgitation may occur at times of high blood-pressur
instance in exertion or in senile arterial plethora, and may di
the valve becoming again competent—as, under treatment or
arterial pressure falls.

Murmurs occurring during diastole may be heard in perica
aneurysm; the former murmurs are not difficult to inter
p. 953).

Dilatation of the aorta is said to be the rule in cardio-ar
generation, the exception (in any considerable degree) in prim
regurgitation. If so, the exceptions are many; I recently
in Cambridge on a case of mere rheumatic aortic disease
there was considerable dilatation of the aorta with "fireman's
dulness.

The association of aortic disease with murmurs simulating mo
those of mitral disease remains to be discussed. The murmur
divided into two classes: those suggestive of mitral regurgitation,
suggestive of mitral stenosis. First, of aortic murmur simulatin
mitral regurgitation four cases were brought forward by Dr. Dic
the meeting of the Royal Medical and Chirurgical Society on
of June 1897. In them, although after death the aort
in each was found to be advanced in stenosis, a systolic mur
heard at the apex, so that mitral regurgitation was either as

could not be excluded. In many such cases, either my own or shown to me from time to time in hospital wards, I have perhaps too promptly and confidently declared my opinion—*quantum valuisset*—in favour of aortic obstruction; I admit, however, that in some of them mitral disease can only be excluded on the principle of "*ne entia multiplicanda.*" Still this principle is a sound one if we do not lean too much upon it. In two cases in which I was led to hazard such an opinion it was borne out by necropsy; there was no mitral insufficiency. In one of these examples the murmur at the apex was musical, and I guessed it to be four to one that a distinctly musical murmur is aortic. In another the murmur was audible an inch away from the patient's chest; it is twenty and more to one that a murmur so audible is aortic. In another again a thrill was perceptible at the base. That such murmurs may be audible in the back I admit,—aortic systolic murmurs often are; but in the cases I have seen such murmurs were not confined to the axillary and infrascapular regions, but were audible anywhere—*passim*, not *ordinatim.* Again, in such cases—and this is true, I think, of those collected by Dr. Dickinson—the arterial pressures were in themselves almost conclusive; the pulse in each was not "mitral," but "aortic," regular and of fair mean pressure. In mitral regurgitation the arterial system is ill-filled, while signs of a rise in venous pressure, cardiac and systemic, are soon manifested. Dr. Norman Moore has suggested that the sphygmograph might be useful in the diagnosis of such cases; for once in a way it might: an anacrotic tracing would settle the question in favour of aortic stenosis, though it might not exclude coincident mitral incompetency of slight degree.

Secondly, an aortic regurgitant murmur may simulate that of mitral stenosis. The murmur of aortic insufficiency generally begins on diastole, is then loudest, and falls as the aortic pressure falls; that of mitral stenosis generally rises up to the systole. Aortic diastolic murmurs in the later part of the pause are very soft, because the pressures in aorta and ventricle are then nearing equality, or have attained it, the vibrations persisting for a sensible moment longer in the walls of the heart. If the murmur be heard at upper and mid sternum, if it begin with the diastole of the heart and taper off during the pause, it is an easy sign to interpret. But if the murmur, not as a rule so harsh or vibrating as that of mitral stenosis, be so soft that it may escape an unpractised ear; if, instead of tapering off from the beginning of the pause, it occupy the middle, or even the latter part of it; if, again, it be barely audible or inaudible at the upper sternum, distinct at the lower sternum, and loudest about the fourth left interspace, the student of the aortic cartilage may be misled by whispers so stealthy and devious. He may attribute the murmur to mitral stenosis; or he may add the case to the list of vanishing aortic regurgitant murmurs, or again he may add himself to the cloud of witnesses to "pulmonary regurgitation." However distinct the murmur may be in the fourth left space, it dies off rather abruptly as the apex is approached.

Finally, the murmur of mitral stenosis may be simulatec regurgitation. There are many cases on record in which a " murmur " was present without mitral stenosis ; in some of thei perceptible lesion was aortic. From what we have seen already (ı student is prepared to understand that murmurs occupying the l or parts of it, not necessarily the initial part, are consistent under certain circumstances significant not of mitral but of aor Against this source of error the observer will be on his guard. explanation does not cover all the ground ; observers of tł authority assure us that a presystolic murmur, heard in the n such a sound as to be characteristic of mitral stenosis, is to b cases which otherwise would be regarded, even on the post-mor as uncomplicated aortic regurgitation. To these cases the late] Flint first drew attention, and his lead was followed by m observers whose records have been well summed up by Dr. I Sansom, who recorded cases of this kind in 1881, has carefully the difficulty again in the new edition of his work ; to this di refer the reader for further detail, as no explanation of the phen as yet established on anything like a certain basis. Sansom a lean to the belief that the presystolic murmur (if it be genera mitral area, and not in the aortic) may be due to impingeme refluent aortic current on the anterior mitral curtain before i taut, whereby either vibrations are set up in the valve itself or ing the valve, the orifice is practically narrowed. Dr. Fisher has two cases of this kind (one of Dr. Hale White's), in both thickened endocardium upon the ventricular septum showed the of the eddy was not in the region of the mitral valve. "The p thrill and bruit were well marked and mitral stenosis was d but at the necropsy the mitral valve was found quite norm aortic valves were healthy also it is interesting to add, and t regurgitation heard during life was due to pouching of the s Valsalva with dilatation of the first part of the aorta." A thir Dr. Goodhart's, is adduced to prove that this presystolic mur be heard in disease of the aortic valve without regurgitation. authors suggest that a meeting of the aortic and auricular curr produce a murmur; if so, surely Flint's murmur should be far mor than it is. One case shown to me in a hospital three years ag physicians as one of this kind, was in my opinion a case of brol diastolic murmur, not generated in the mitral area at all. T no rumble ; the murmur was audible to left of the sternum in the scapular region. Still rumbling presystolic murmurs, w do no doubt occur in aortic disease unaccompanied by mitra All I can do then is to warn the reader of this source of eı that murmurs form but a part of cardiac diagnosis. It has t for us that these invaluable aids to diagnosis have received ev proportionate share of attention, but it has been at some loss of ı of other aspects of cardiac disorders, some of which are of no le

Pain.—Distressful sensations of the nature of pain are more common in disease of the aortic area than in other diseases of the heart. The distress may range from a slight oppression to breast pang; while a fair compensation is maintained, and there is no active aortitis, no discomfort may be felt, otherwise the distress may become agonising and almost constant. Its form is that of angina pectoris. When the insufficiency of the aortic valve is of acute onset, as in sudden rupture, the pain and oppression may be very great; but unless the mischief be of extraordinary severity—bad indeed almost beyond hope—the pain will pass off as the inflammation in the area subsides, the reserve capacity of the heart comes into play, and pressures are readjusted: thenceforth, until the organ begins to fail, discomfort may be absent—at any rate in patients under middle age. If it be in elderly persons, the subjects of general arterial disease, that angina pectoris in its major or minor forms most frequently occurs, it is by no means confined to them. I recently witnessed a very distressing and persistent angina in an undergraduate, the subject of recent rheumatic disease of the valve, and probably of aortitis. A sense of substernal oppression is the first hint of it; it is felt on distension of the stomach and bowels, and on ascents, even the gentlest. In extreme cases, or in persons of the peculiar temperament which favours the phenomena of angina, assaults of this kind may come on during complete rest, probably in obedience to unseen tides of blood-pressure. That angina pectoris comes on during effort only is a false aphorism based upon too smart a description of such cases; not infrequently it comes on even during sleep, adding a new torment to the bitterness of death. Of muscular movements those of the arms seem to be the most efficient in producing anginose pains; it has been stated that movements of the arms are the most instant in their effects upon blood-pressure. Another surmise is that persons of a gouty habit are peculiarly liable to anginose attacks and complications, an opinion based upon no little clinical experience; it is but a part of the truth, however, as angina is even more tyrannous, if less lethal, in persons in whom the neurotic habit is conspicuous. Such sensations are not so common or conspicuous in aortic stenosis as in regurgitation. As the aorta is probably the seat of them this distinction is intelligible. Another seat of pain in aortic regurgitation, and this too rather in the later phases of it, is gastralgia, or a suffering so described. This pain is to be discriminated from the aches, severe and trying as they often are, which seem to have no deeper source than the intercostal and neighbouring spinal nerves. With the gastralgia is often associated the persecuting flatulence which besets all cardiac affections, even the functional. To belch up wind is attended with relief, but it is another thing to say that the wind is the sole cause of the distress, and it cannot explain the recurrence of the "gastralgia," which I suspect is allied to angina.

It is alleged that there is some connection between tabes and aortic disease. Ruge and Hütter found aortic disease in nine cases out of 138.

of tabes (6·5 per cent). In only one of these was there no pro syphilis, and in five this antecedent was definitely ascertained. rheumatism counted for very little. Sir W. Gowers accepts the as a causal one, and Grasset and Rauzier are of the same opin probable explanation is that both diseases belong to the syphil and may be associated in young persons before the ap senile atheroma. Other authors regard the connection as one coincidence. No confident opinion can be expressed at prese may be that in many cases of aortic regurgitation the gastralgic are directly of tabetic origin. How often do we wish our again for better investigation! It is but the other day, af completed an examination and discussion of a case of thoracic that my colleague in consultation was wicked enough to tell not found out that the patient was tabetic. Though the gait w affected, I had to admit, when told, that such was the case. I syphilis was no doubt the nexus, and an insidious tabes m origin of some symptoms not directly attributable to the car disease.

The nervous system.—Besides pain, which strictly speaki come under this head, there are other nervous disorders which marked in aortic regurgitation than in other forms of cardiac di an article on cardiac delirium, published many years ago, I the sufferers from aortic disease show an occasional liability t derangements. Even in the latent or stealthier phases of sufficiency we may note more especially certain mental pert which are not unknown in other heart diseases. We note a re a fretfulness, a change in temper amounting sometimes, as th advances, to violence; in rare cases the restlessness sometimes g as to urge the patient to spring from bed, to perambulate the even to jump out of the window. We may compare the deliriu cases of aortic regurgitation to that of alcoholic pneumonia; these extreme degrees it occurs chiefly in men, it may be so tr as to make a male attendant necessary. That it is not al proved by its outbreak or persistence in patients who are been under continuous observation and restriction. Much of lessness of the delirium is due to the fact that it is usually a of place: the patient is under the delusion that he is in a stran or far away from home; pacified for a few minutes, or for a few delusion seizes him again and again with an agitation which i with the worst consequences to the cardiac disease. Prof. C makes a like observation. The association of insanity with cardi has been studied by Mickle, Ball, Fauconneau, and others. A mental disorder, headache is frequent in aortic insufficiency; and dizzy sensations, momentary obscurations of consciousness, twit even convulsions, may indicate the perturbed conditions of the functions by way perhaps of the circulation. The vascular in is perceptible to the patient whenever he stoops. Sleeplessnes any means always due to cardiac uneasiness, is often very troublesome, and is especially noticeable in aortic insufficiency.

Nutrition.—Although the arterioles cannot be contracted, as sometimes alleged,—or we should not see the capillary pulse,—yet pallor and some falling off in flesh mark another distinction between aortic insufficiency and mitral disease, in which the face is congested; and emaciation, if present, may be concealed by venous turgescence or arterial œdema. So long as dilatation of the left ventricle is compensated by hypertrophy, so long as the cardiomotive force keeps up, there is practically no anasarca or ascites. Filling of the pleural cavities, swollen legs, albuminuria indicate a slackening ventricle and increasing residual blood; the heart is entering upon that final phase of demolition which has been described under the diseases of the myocardium, and must not detain us here.

Respiratory system.—While the mitral orifice and the myocardium are sound the pulmonary circulation is protected. It is in the final stage of a shattered heart that the bases of the lungs begin to fill. These changes often appear before there is definite evidence of mitral insufficiency—at any rate before a murmur is generated, and even before the extension of dulness over the right ventricle. As the ventricle is distended the papillary muscles may fall relatively short; or in some other of many ways the mitral machinery may be deranged: yet even with a competent mitral valve, as the residual blood in the left ventricle becomes more and more, and the regurgitations perhaps larger and larger, the arterial head will dam back the venous. When a murmur of mitral regurgitation appears the end is not far off; and therewith the case travels out of my sphere.

Dyspnœa is scarcely to be called a prominent symptom till this last stage is reached. The dyspnœa of the earlier stages is rather an inexplicable perturbation which the patient himself can hardly describe, and which, if an exact person, he usually declines to call shortness of breath: he speaks of it rather as a sense of oppression which impels him to sit up; it partakes of the nature of angina. Nay, often, as in angina, he may evade a strong inspiratory effort. At times, however, and in later phases of the disease, the patient may be seized with "cardiac asthma," when the gasping and shortness of breath are distressing. Still, this is not quite the panting of mitral disease: the excursions of the chest are less conspicuous, and have more of a nervous or spasmodic character. It may be a call of the bodily tissues upon the heart for more blood, a call not so much for the "respiratory pump" as for more driving power; or, again, it may have a toxic origin. If expiration becomes audible a little distance away, and both inspiration and expiration assume a tubular quality, such as horsemen call "roaring" or "whistling," then, however slight this may be, the trachea is so far constricted by a dilating aorta.

Cough is often present—generally indeed—and may be an intolerable evil. This cough, when it does not spring from incidental causes, is due to pressure of the dilated aorta, either directly upon the trachea, or upon the laryngeal nerves. Unless there be some contingent catarrh there is no expectoration, or no more than is hawked u cough. In cases of considerable dilatation of the aorta the c be of frightful severity. One patient of mine, when he felt coming upon him, used to throw himself on his hands and k such sufferers will anchor themselves to bed or table to mit racking of it.

Sphygmographic signs. — The ordinary tracings which a books and essays are of little worth. The more valuable c as those of Mahomed, Galabin, Riegel, Lorain, and others some points of interest. Hundreds of tracings are publish prove no more than the inadequacy of the sphygmograph t the finer components of the aberrant pulse. It is charact the tracings in aortic insufficiency to show a hook or "cr the summit of the percussion wave which, in aortic reg with a strong ventricle and little or no aortic obstructi course very high. The sharp return of this "hook" is said t the rapid arterial recoil; but to my eye, like many other suc it exhibits nothing more than the inertia of the lever. No waves due to this cause are too often interpreted as records that secondary vascular wave. After a sharp fall of the lever regurgitation, or in other states in which arterial resistanc a second wave of inertia may also be seen, and even a thi a tracing recently published and elaborately explained. Su mean nothing more than the bouncings of the long and li after strong percussion. It is remarkable that the dicrotic w persists (Fig. 60, p. 937). Now if the dicrotic wave be due to the aorta, we might expect that when the bottom of this knocked out this recoil would be prevented; but this is by always the case. Dr. Samways urges that the dicrotic wave the longitudinal recoil (shortening) of the first part of the a may possibly explain the persistence of the wave under the circ we are considering in any case stenosis would promote it. probable that as regurgitation increases the dicrotic wave obliterated; but it does not appear that this indication has any i prognostic value. The presence of more or less stenosis migh cated by an anacrotic wave in the tracing. It is difficult to precise conclusions from the sphygmograph as to degrees of a the tendency in such cases is, of course, to a broader-topped wa sphygmometers of Hill and Barnard and of Dr. George Oliver se to take a practical shape, and, if so, mechanical aids of great be placed at our service. By such means many difficult probl obscure, will be made clearer to us.

Diagnosis.—Much has been already said indirectly in thi In cases of uncertain diastolic murmur the absence of thrill c tribution about the base, the absence, in the earlier stages, of first sound of mitral stenosis, of reduplicated sounds, of eviden of pressure in the pulmonary circulation, and constancy of m changes of position, will indicate that if there be a murmur in the mitral area also, it is but the flapping of the upper limb of this valve (Potain and Sansom). I repeat that in following a murmur from apex to base, it may not only go underground for a space, but also may emerge with a change of quality; and that murmurs of aortic regurgitation may be exceedingly distant or faint, may frequent strange quarters of the cardiac area, and may be inaudible at the aortic cartilage. In the last stage the failing systolic sound is as short as in mitral stenosis, and the liver enlarges and hardens. The jerking of the arteries too may then subside, and the case becomes virtually mitral. Duroziez's sign may be useful, but is hard to make out in an œdematous thigh.

In a patient, whom I saw but once, I had some hesitation at first in deciding whether a chafing diastolic sound at the base were due to aortic regurgitation or to the pericarditis of chronic renal disease. A study of the whole case, however, left no doubt of the latter interpretation.

Prognosis.—The course of aortic regurgitation is towards death. As in all heart diseases the main factors in prognosis are four: the age of the patient, his calling and habit of body, the kind of lesion, and the degree of lesion. An accurate knowledge of the history of the patient and of his symptoms is very important, but it is not always to be had. I have a difficulty in recalling cases of mere aortic regurgitation in children; such cases, if rheumatic, have no doubt a long average survival. A deformed valve segment must in all cases be a strained segment, and meet for chronic inflammatory and atheromatous degenerations. A clean rent in a healthy valve segment should be a less destructive process than a lesion of equal degree due to atheroma; it is said that a clean rent in an aortic cusp has been known to heal. As age advances the prospects of the duration of life grow less and less; the lesions may be worse in kind, certainly adaptation is less ready. In atheroma aortic regurgitation signifies not only progressive disintegration, but also an accelerating rate of it, and prognosis is graver with regurgitation than with obstruction: aortic direct murmurs being, as I have already said, the ordinary feature of atheroma, regurgitant murmurs the extraordinary. Death may suddenly intervene in the period of latency, primary or secondary, but the period is one of comparative safety; when the attention of the physician is drawn to the disease by complaints of retrosternal oppression or of uneasiness on ascents, the stage of dissolution has begun; whether the origin of the mischief be in old or young, in strain, rheumatism, or atheroma; though in this last kind dissolution may be more rapid. In strain such sensations may be felt at first before re-adaptations of cardiomotive functions have become established; but if the patient's life is to be a comparatively good one they should pass off for some years, as the reserve capacity of the heart comes into play (secondary latency). The patient may go about his work again in ignorance of the fatal rift; yet, when he is brought up, sooner or later, by some uneasiness about the heart, he does not forget to tell the physician how that on a certain occasion of effort he felt a strange and distressing sensation in the heart. This event may have been five years before; bu it is not more than two or three, and may be much less.

The duration of the latent period—primary or secondary- more on the degree of insufficiency than on the soundne cardio-arterial system: for, unless it be in the case of syphilis undermined by atheroma are withdrawing on account of virtual heavy work; and if in older men the conditions of nutrition m favourable, those of labour are less exacting. If, however occur in a man whose arteries are degenerate, the latent perio brief. In such a case, recently under my care, the consequent of disease never receded at all. When we turn from ruptu sufficiency gradually established, we find, as I have already said literary a view of the matter is taken by many writers, especial division of chronic aortic disease into the cardiac and the cardi A long survival is not unusual in cases of general cardio-arteri in elderly persons, while on the other hand "young cases" poorly, and last for a briefer span than we had anticipate the duration of a heart maimed by aortic insufficiency may be as short in young persons as in the old and atheromatous granted in respect of younger subjects in whom the invasion o is unchecked; it is not usually admitted of rheumatic diseas this process consists in a proliferative fibrosis which, as oppose "replacement fibrosis," too often has ruthless cicatricial cons Healthy as, apart from the local disease, the heart and arte otherwise be, the progress of such cases is often inexor were paradoxical to say that the outlook may occasionally in cardio-arterial atheroma, but the part may not be far whole truth. I have said that the capacity in elderly perso fairly sound hypertrophy of the left ventricle is usually much un even in the presence of dilatation of the aorta, and of stiff vessels, machine with a fair muscle at its centre may last many unless one or other coronary artery be blocked; or miliary a form on the cerebral arteries. Let sanguine prophets say w may, ten years is a long time in any case of aortic insu and, given equal degrees of insufficiency, I would not despair respite in temperate and tranquil elders, until they "be gathered, not harshly plucked, for death mature." Every p experience must remind him that to be "harshly plucked" is fate of the older of these patients only; of young men who die no small tale die of aortic insufficiency; and to die of sync a sound or fairly sound heart muscle happens to old as w young patients. To say that the disease in the cardio-arteria "progressive," and in the rheumatic or strain cases not necessarily academic a distinction, and untrue even as that: aortic insufi always "progressive," even if the local disease is not. If the be asserted it is because observers are in a state of reactio the black prognosis of all and any heart disease which prevail our fathers; now we are in the opposite extreme, and are buoying up our patients with too crude a hope. Some young patients die unexpectedly soon, some old ones live beyond expectation. Mitral insufficiency is the only heart disease which, under favourable circumstances, can be nursed to an indefinite duration. I have now under my occasional observation persons still leading useful and active lives who have lived a quarter of a century and more with mitral regurgitation; but I cannot remember the survival of any patient with aortic regurgitation for fifteen. If the patient, whether in a palace or in a workhouse, be a man of easy circumstances and tranquil occupations, he has the greater chance of survival. Care or worry, bustle or toil will kill him. There are men of such a temperament that they cannot form sedate habits: recklessly, as it seems to the doctor, they skip up stairs two at a time; they puff after trains; they climb over five-barred gates; they bounce up from deep sleep to pass water, and so forth: they do not mean to run these risks, but such is their incorrigible temperament. With such persons discipline must be attained by spending day after day in drill, in gaining self-control, in repressing volatility. In this precaution there is nothing false to a man's best self; it is the way to get the most work out of himself before he dies. Persons in toilsome callings must change them; and spend the perhaps no less useful remnant of their days in some easier duties. Due vigilance may be exercised without the encouragement of hypochondria; as some one well put the rule: find out what you can do, and do it; find out what you cannot do, and never do it. The conditions of survival are more favourable in women than in men.

The big ventricle, efficient as it is, racks the machine from the beginning; the aorta, being of elastic tissue and not of muscle, suffers under the thrust, and the means of the heart's nutrition, instead of increasing as demand requires, are gradually sapped. The watchful physician may then note that muscular effort no longer raises, but even reduces the blood-pressure—a bad sign indeed.

Anginose pains are always menacing in regurgitation, yet even they may be kept at bay by the nitrites, it may be for a year or two; but the respite is a life of troublous days—a life of pain, of slavery to drugs, of bitter physical and mental adversity. Anginose pains seem to signify less imminent danger in women than in men, although in men they are more common; this, if true, may depend on the greater docility of women under treatment. Sometimes angina appears only for a time, with a push of aortitis.

In insufficiency death is not always sudden; some patients drink the cup to the dregs; life is protracted from phase to phase of cardiac disorganisation. Usually, however, the thread of life is snapped before involution is complete, before these later stages of cardiac dilatation and rise of venous pressure are accomplished. Such patients sometimes die of asystole, far more frequently they die of syncope: the heart, not yet quite played out, comes to a sudden stop, probably under some reflex interference. Although then the signs of cardiac dilapidation will be

noted with apprehension, gradual dissolution is often avoid
seeming caprice death cuts the thread after rather too good a
quick step into a railway carriage, or a start up from bed ; or
bolt may be mercifully drawn during sleep, and the last year
a life may be happy even in the ending of it ; for as Bacon says
times death passeth with less pain than the torture of a limb
most vital parts are not the quickest of sense."[1]

To enter into a discussion of combined lesions of the he
lead to repetition of the work of other contributors ; but it
needless to say that in every estimate of the duration of life
insufficiency the values of the other component parts of the h
be estimated : such estimates are to be found in the chapters
diseases of the organ. Again, it is of the first importance
whether a coincident lesion elsewhere, valvular or muscular,
pendent or dependent on the aortic. It is contrary to my exp
assert, as many have done, that coexisting mitral regurgitation
in any stage of aortic insufficiency, except as a relief to the ao
case of angina ; that moderate mitral contraction may be so i
able. In rheumatic cases, aortic disease usually means a more
cardiac damage, and in this respect again the prognosis is worse
than in mitral insufficiency.

That "apex murmurs" are often mere aortic direct murmu
said already. Loudness of murmur, other things being equal,
favour of sustained cardiomotive force, and, although a murmu
the point of indistinctness may be consistent with slight or
injury, on the other hand a murmur may wane with the hea
generates it. A quickening pulse is of ill omen ; if not due to t
causes, it means a larger residuum at each contraction and
arteries, as tested by raising the arm. We are told that a fa
specific gravity of the blood is likewise of ill augury. If stenosi
with insufficiency the peripheral arteries will be the less in d
moreover, in stenosis they contract upon their contents, in regu
they are slack. Increase of the area of cardiac dulness vertic
be a good sign ; its increase transversely is a bad one ; and,
generally, changes in the chambers are of far more importa
changes in the murmurs ; as we have seen there is an element o
in murmurs, which may rise, fall, split, or perhaps vanish for
without definite prognostic meaning.

Of intercurrent diseases the infections are the most injurious
effects upon the lame heart ; and of these influenza and diphth
the most malignant.

If possible "functional" perturbations of a transient kind
distinguished from changes in the myocardium ; but to estimate
of the myocardium in fairly stable cases of heart disease is very
The results of treatment, especially in the use of digitalis, perh
give us some hints of this kind. Arrhythmia, alteration of othe
diminution of urine, the appearance of albumin or hyaline cast

of remedies previously effective, are of sinister meaning. Neglect of treatment until late in the disease is against the patient's prospects; the command of skilled treatment and the means of carrying it out are in his favour.

Mitral insufficiency is not infrequently cured; aortic never. As in Hermann Weber's case, though the murmur may cease, the mischief stealthily advances, and may bring down the stricken man when he least expects it.

No error is worse than false precision; none more gratuitous than prophecy: still in human affairs we cannot get beyond moral certainties, and patients or their friends often demand of us a fallible prediction. Given a moderate lesion and good conditions within and without, I should say that in a patient under five-and-thirty years suffering from rheumatic, syphilitic, or traumatic aortic regurgitation, the prospect of life is about ten years; rarely more than twelve, save in cases where the lesion is nominal in degree. In persons over fifty, in whom the arteries are atheromatous, and the aortic insufficiency a later stage in the work of decay, three or four years may be expected. On the other hand, if the aortic insufficiency be an early sign of atheroma about the base of the aorta, and the patient in easy circumstances, death may be kept at bay for six or eight years. The previous rate of change in the individual is of course a most important element in our judgment in each case. In obstruction alone the expectation is much longer. If in aortic disease, even at this later age, the lesion be syphilitic, as in a case now under my occasional observation, the prospects are much better; by careful treatment even ten years may be added to the sum of days.

Cases of alleged recovery from aortic regurgitation are recorded from time to time. I have said what I think of such stories: the patient was not watched long enough; murmurs may be evanescent, not so the lesions they signify. No less an authority than Leyden, however, has recorded such a case of recovery, but after a traumatic lesion (51).

Cerebral embolism is prone to occur in aortic disease of whatever kind; pulmonary apoplexy occurs, but does not take the place it does in mitral disease, for obvious reasons. For an account of these events the reader is referred to the chapters which deal with them.

Treatment.—Give your prognosis on the best suppositions, treat your patient on the worst. The treatment of aortic disease, and I now imply both kinds of it, falls into the natural divisions of diet, management, and drugs. In *diet* we have to look to three points: to the sympathy between the heart and the stomach, to good nutrition of the heart, and to moderation of its work. We must avert indigestion, and administer nutritious food without either raising the arterial resistance or increasing the heart's output. Indistinctly we are aware that there are diets which promote arterial resistance, and so far as our lights go we must elude this danger. Many of the elderly sufferers from aortic disease are gouty. In such persons we should avoid all that encourages this habit (vol. iii. p. 187). On the other hand, to reduce the diet below the needs even of a person who can take little bodily exercise n us into the peril of pining the diligent heart; and to excl genous food in order to avoid goutiness may throw the patie diet of carbohydrates, a diet both bulky and provocative of and gastric acidity. As indeed in gout itself, a careful mixed answer best; and on two points we must especially insist—c tion of liquids during meals, and on thorough mastication of whether it be soft or hard. In more than one case I have s relief to follow fine chewing and the restriction of liquid Even between meals it is not well to allow the patient to drink the blood-pressure can hardly thus be raised, as Huchar enormous quantities would be required for such a result, but t of the ventricle may be increased, and therewith its work. It i needful to insist upon the use of food which is at once easy and worth digesting; at the same time some foods are indirec eating if they are grateful to the eater, and thus stimulate the s

Alcohol is overdone in all heart diseases. The immediate rel sufferer is often considerable, and as a cardiac stimulant in time it is indispensable. As an ordinary article of the patient's con its use is not without some drawbacks; it disturbs blood-pre effects accumulate more rapidly for harm in persons who cannot t exercise, and the perpetual nips, in which too often they are led t themselves, directly induce those very conditions of venous stagn degeneration of the cardiac muscle which we are on our guard On the other hand, such patients are often cheered by a litt and water, a light hock or some well-diluted spirit with mea being strictly reserved for critical occasions. If on every palpitation or faintness the nurse is to run for the brandy b patient's state will grow worse rather than better.[1]

In respect of management it is difficult to give general direct no cases are tact and experience more valuable. The young pra must remember that if, on the one hand, there be a danger of inj the effects of a careless life, on the other the harmful effects of "v arianism" are no less; and the patient in gaining his life may We must trim our treatment according to the phases and pec of the individual. Fraentzel well says that to know that one h disease may be more mischievous than the disease itself. Let you understand that he has a weak heart, and that he must rigidly your rules of life, but not otherwise fash himself; and to some and trustworthy friend of his tell the whole truth and the risk o death if such there be; that like other wise men the patient n his affairs in order.

In the matter of *exercise* often lies the decision whether th be allowed to follow his calling. If the occupation be one of labour the patient cannot but leave it; a working-man must seek some quieter means of subsistence, as a caretaker or the like. A sportsman must contract the field of his pastimes: the salmon rod must give way to the lighter engine of the trout-fisher; cricket to golf: the moors must be forsaken for the stubble and the covert, and the hunter exchanged for the nag. Cycling is by no means an unfit recreation for the subject of heart disease, in its earlier stages; if so be that he is already a good rider, and will ride circumspectly. Whatever pursuit be admitted, and much will depend on the degree of incapacity, one caution must be remembered on all occasions, namely, that although the sense of oppression which checks exertion can be "worked off," unless very severe, by perseverance, it is a grievous error thus to persevere. It seems then that the heart does not so much pull itself together, as become blunted to the persistent strain. The cry of the burdened heart must never be disregarded. And yet again while we offer this necessary caution we shall not forget that perpetual timidity is even worse for the patient than occasional indiscretion. So long as he lives let him live, so far as may be, the life of a good citizen. Above all do not let him mope, or become entirely possessed by the blind and ignoble desire of the mere prolongation of days. We who have to minister too often to these unprofitable uses of the world, can proudly point to men, great examples in our own profession, who showed us how to live most nobly when death was treading in their footsteps. The physician who inspires moral health into his patient brings comfort also to his body.

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[1] In his work on "Senile Heart" the veteran physician Dr. Balfour gives directions for treatment of heart disease, and at greater length than it is possibl give in this place.

Drugs.—During the latent period of aortic regurgitation those drugs only will be required which are of service in common ailments; specific remedies are rarely necessary. In case of acute onset, such as rupture of an aortic cusp, the measures described already under the heads of management and diet may be all that is required. Hearken, let us say again, to the cry of the burdened heart; no hypertrophy can go forward while the organ is embarrassed. Under the unwonted stress it may be necessary, while the heart is pulling itself together, to put the patient to bed until the heart has begun to turn its reserve capacity into the statical condition of hypertrophy. As this is attained the patient will return gradually to the ordinary habits of life. I have not found digitalis of great service in this stage. On the contrary, gentle mercurials, gentle salines, a little potassium iodide—means which reduce blood-pressure—are more helpful. In this stage too much care cannot be given to save the work of the heart in all directions, whether of muscular work, of the digestive and other organic functions, of cerebral and emotional activity. When this stage is passed, and something like compensation established, the patient will betake himself to moderated exercise and a more bracing moral life. If during the early period there be intercurrent times of strain, due either to indiscretion or to some fluctuations of inner health, intervals of more or less seclusion will again be enjoined, and the above indications repeated. The best all-round medicine for heart disease in these phases is blue pill.

In preparing this section I saw before me the duty of readir multitudinous arguments which have been written upon the use in aortic insufficiency, a grievous prospect: this intention I l up. After all that is written, the subject lies in a good deal logical obscurity, and it is best for the present that each obser give the results of his own impressions as simply as possible. its use in aortic insufficiency we have the eminent authority of in favour of its use that of Balfour.

Let me repeat that if the excised heart of a small animal be to a pressure bottle, that pressure can be increased gradually seen that with each increment of pressure the base line of the car curve will fall; the ventricle dilates. Why does not the vent cases, in health or in disease, dilate to its extreme limits at once of its "tone"; probably also because of the well-known react "loaded muscle." A loaded muscle, although prevented from lever so high as before loading, contracts more strongly. Fo know of tone, a property of the highest importance in cardiac we are largely indebted to Dr. Gaskell. My own view is t property of tone be fundamentally one with that of contr has become so far differentiated from it that the two virtues n cussed separately. Tone we may define as that property in he or other hollow viscus which preserves the mean diameter of contraction as that which enables the organ, nevertheless, to obe and to perform particular acts. The vermicular movements of and of an arteriole are due to the quality of contractility; preserves their mean diameters in spite of distension or c Were it not for tone a hollow organ, often subject to extravagant would be strained and perhaps ruptured. In the heart it is t does much, if not all, to prevent loss of form under the great of internal pressure.

In the year 1868, when Dr. Milner Fothergill was th medical officer to the Leeds Dispensary, I placed a large col cases of heart strain under his superintendence, and in ord our remedies for these patients, we carried out together a experiments on digitalis, which Fothergill afterwards publis Jacksonian Essay. We demonstrated the effects of digitalis on of frogs and small mammals, effects which are now too well need narration here. Suffice it to say that the chief effect is a of tone, which may be pushed to a degree inconsistent wi function. When a solution of digitalis is dropped on a frog's see an increment not of contraction but of tone. The h on contracting with a smaller and smaller volume till for lack the animal is moribund; when other variations, such as fibrilla tion, may supervene. In aortic insufficiency, the regurgitant st not exactly "impinge upon the inner wall of the ventricle at of relaxation," for it can scarcely be said that the ventricle is " the mischief is that the pressure is abnormally increased at when the muscle is at the disadvantage of greater cubic capacity, and when the direction of motion is with the regurgitant stream. The "loading" indeed, if not excessive, stimulates the organ to stronger contraction (reserve capacity), and this dynamical reinforcement becomes statical as hypertrophy. The same process being repeated again and again, the heart attains the huge dimensions with which we are familiar; and in the muscle itself there may be no limit to such increase, the limit being imposed by the scale of the associated structures. Were tone absolute, there would be no dilatation; hypertrophy alone would take place, and the output would be too small; on the other hand, if, as in chronic strain, the tone is overborne little by little, dilatation ultimately soon surpasses hypertrophy.

Tone, then, is the quality to be watched and supported; and in digitalis we have a means of intensifying tone, of moderating distensibility. Now tone, like any other quality in excess, may be injurious, and the output of the constringed ventricle may fall short of the demands of the system. That the residual blood should become less and less after each contraction, and possibly vanish, is good; but if the shrinkage of volume goes on farther, the output may fall farther below the needs of the system than it did in the case of excessive residuum. On the body the result is practically the same. Hence one chief reason why digitalis should be used with especial precautions is lest diminished capacity come to the same thing as asystole. Again, when the muscle falls into degeneration digitalis seems to have other injurious actions, the nature of which is obscure. We cannot get fatty hearts of frogs for experiment. It would appear that digitalis acts not only on the tone of the cardio-arterial muscular coat, but also on the vagus. In tachycardia, and other conditions of rapid pulse, digitalis by giving tone to the arterial system often causes diuresis without reducing pulse-rate—without, that is, being able to get a hold on the vagus. Conversely in a degenerated heart digitalis often seems to stimulate vagus action while tone is failing; then we get slowing, coupled beats or intermittence of the heart without diuresis (Fig. 61). Indeed the vagus interference by slackening such a heart may conspire to its further dilatation with increase of residual blood. This perilous result of digitalis is but too well known to us not only in aortic regurgitation, but also in other kinds of failing left ventricle. If, then, diuresis do not soon appear, the drug must be stopped and a little alcohol substituted for it. So long as the cardiac muscle is in fair condition, the working of digitalis counteracts the distension of the left ventricle and lessens the volume of residual blood, an aid too valuable to neglect if contingent dangers, such as an undue reduction of output, vagus meddling, or oppression of degenerate muscle, can be avoided.

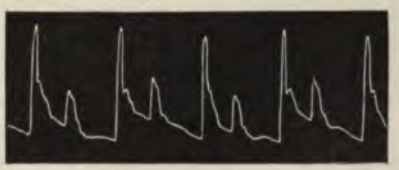

FIG. 61.—Aortic incompetence with bigeminal pulse due to action of digitalis. (Graham Steell.)

If we can use the drug so far as to pull the heart togeth
constricting its cavity or arousing the vagus too much, we
ground so long as the muscle is sound. Now we find, prolong
or not, that in practice digitalis, used with discretion so a
the heart and not to string it up too tight, is indeed the
able weapon in our armoury while the cardiac muscle is so
advent of degeneration of the muscle cannot well be detecte
administering the drug experimentally in single doses, say in
10 minims of the tincture once every second day, taking t
urine as our guide. As to the "prolongation of diastole," in
propulsion is better, refluence is less; in so far as the ventric
is less, residual blood is less; moreover, the pause is not all
stole; during it the pressures in aorta and ventricle approxi
during the later part of it are indifferent, or even reversed
acceleration of the blood is almost entirely an abbreviation
stole, yet acceleration is not a help to the heart, but a sign of it
The organ is then dependent for its integrity on its tone, an
have seen, by digitalis the residual blood may be reducible b
constriction of the ventricle, the abnormal pressure at the fi
the diastole, when it is highest, tells upon the walls at a mom
cubic capacity, and at a moment of greater resistance. In
the ventricle dilates, the output, other things being equal, re
stant, and the mass of residual blood increases; if by digitali
be enhanced, output and contraction volume will approximate

What are, then, the rules for the administration of digitali
insufficiency? No one would give digitalis when a big heart i
ing along its course and the arteries bounding under its puls
the left ventricle be relatively too capacious, and the apex be
diffused, put the patient to rest with his feet up, so as to dimi
pressure; and put him on tender meats, avoiding much carboh
much liquid. Gentle deobstruents will probably be required als
under these means the symptoms and signs of dilatation co
minister one dose of digitalis, and if it is at least harmless,
another twenty-four hours later, noting the rate and rhythm o
and the volume of the urine; thus watchfully a safe judgme
made as to the further use of the drug. Although a pulse ov
suggest, it does not dictate the use of digitalis; some evidence of
is required; on the other hand, it can rarely or never be we
digitalis if the pulse be at or below 75. I have a prepossessi
digitalis in any case in which the heart intermits: it may be
justifiable to give it in cases in which the intermission is but a s
element in a rhythm otherwise quick and irregular; but if inter
the sole or a leading feature the drug is better avoided. If in l
the right side of the heart seem disturbed, digitalis can rarel
wise than helpful. In such cases, indeed, we do not look to
to murmurs or even to valves; we watch the apex beat, t
cardiac dulness and the volume of the urine.

Perhaps digitalis is least needed in aortic stenosis; yet even in this malady, if the stenosis be constant or increasing, and the heart yielding, it may be necessary to introduce it occasionally, remembering, however, that, if the obstacle in front be very great, to spur on the heart is to ride for a fall.

The preparations of digitalis are so many, and the advantages and the drawbacks of this and that are so many, that I must refer the reader to works on pharmacology for full discussion of these very practical points. In a case to which I was called in consultation three or four years ago, Nativelle's granules were used by the medical man in charge of the case, one of dilated heart; these proved so helpful that I have prescribed them occasionally since that time, and certainly can confirm my friend's good opinion of the preparation. I see Dr. Balfour also uses these granules, or a syrup made by Nativelle. I believe in the "cumulative action" of digitalis, but have no notion in what it consists; whatever preparation be used, it is well to use it intermittently. Death is so often sudden in aortic insufficiency, that its occurrence during the use of digitalis, or of any other means, must not be attributed too readily to medicine. If the stomach be disordered, digitalis, if given at all, must be given subcutaneously.

Strophanthus is sometimes of great service; more frequently it disappoints us altogether. I have little experience of it in aortic disease; generally speaking, I should say that it is much more useful in young than in old people; it is in patients under thirty years of age that I can recall many cases of heart disease, chiefly of mitral regurgitation, in which the drug acted with celerity and efficiency.

Arsenic and strychnine come to our assistance at times when drugs which should be more directly potent fail or are inadmissible. If strychnine be prescribed at a critical moment and rapid effects be desired, doses much larger than those regularly given are required. For an adult fifteen drops of the liquor are not too much thus to prescribe as a single dose. If the patient complain of some slight rigidity the dose is intermitted, and no harm comes of the reaction. Arsenic is more adapted, of course, to chronic medication, and, whether as a nervine or muscular tonic, is very useful. Sir William Broadbent, I see, regards the virtue of phosphorus as even superior to that of arsenic. Caffein—the pure caffein of Merck, not the citrate—is an old ally of mine; it stimulates the heart when it flags, and it promotes diuresis. It is also useful in "cardiac asthma." From 1 to 3 grains may be given for a dose; and in some persons it is better to push the drug early in the day, pretermitting it of an evening lest it disturb sleep. Caffein is useful as a cardiac stimulant in cases of slow pulse in which digitalis is out of the question. Good and strong coffee taken black may be substituted for the caffein if no great precision of dosage be necessary.

The nitrites are perhaps never required until symptoms of an anginose kind arise; then they are invaluable palliatives. The researches of

Professor Bradbury and Dr. Marshall indicate that of these a erythrol tetra-nitrate is the most useful, as its effects are eas lated and more persistent. The amyl nitrite, being the most action, is to be preferred at critical moments; but its effect is In cases of severe angina these agents are very precious to us, by relieving the stress upon the aorta wherein the pains orig end attained by slackening the heart as well as by expanding pheral vessels. I cannot but suppose, however, that these ag some anodyne virtue besides the mechanical, for I have see relieved by a nitrite, while my finger was unable to detect a in the blood-pressure. In extreme cases of aortic disease the a angina may be so frequent that the life of incessant suffering a hension is almost more than can be borne; in these cases the nitrite of amyl may become almost a slavery. A craving seems up which is not easy to discriminate from the sinking of the ang Bradbury and Marshall have made researches into a method o ing the use of vaso-dilators with digitalis which seems to b siderable promise (48).

I suppose that chloral is a dangerous remedy in heart especially in degenerate heart. Sir W. Broadbent proscribes it al Dr. Balfour, on the other hand, speaks of the drug with ap When chloral first came out, being less troubled with modern sp about blood-pressures than we are now, and undisturbed by G Shore on chloroform, I used chloral freely in the restlessness diseases, not excluding those of old people. Indeed, to many with degenerate hearts I gave the drug year after year, and with the greatest comfort. The anxious, perturbed nights sufferers are full of trouble and peril, and sedatives cannot be I now use chloralamide, which, I am told, is safer than ch certainly it acts well, though scarcely so well perhaps as t Balfour, while clinging a little to chloral, suggests the use of or paraldehyde instead. Trional is perhaps better than sulph neither is so useful as the drugs just named.

It is now thirty years since, in the third volume of *The Pra* recommended the hypodermic injection of morphine in heart dis testimony of the best kind, such as that of Dr. Balfour, has my advice. Dr. Leonard Hill says "morphine is one of the constrictors and cardiac tonics we possess." By the mou is behind other sedatives in value, its use being attended drawbacks; but hypodermically, in doses beginning at one a grain and gradually ascending to a quarter of a grain if it is a precious means of relief. The physicians who still prote its use are unfamiliar with the practice. There is no remedy w forth so warm a tribute from the patient himself, who, aft of watching and agony, sleeps a peaceful and natural sleep, a almost forgetful of his plight. Of the drawbacks to the c use of morphine I may refer to the article on the subjec

p. 887). Like any other potent remedy, it must be used seasonably and discreetly.

Ammonium bromide is sometimes of service in the minor degrees of restlessness, but, if long continued, is apt to be depressing. All the salts of potash are to be avoided, even the nitrate. Convallaria, sparteine, cactus, and the like, are only known to me in the blind uses of despair.

In this section I have spoken almost entirely of the treatment of insufficiency; of stenosis, I need not say more than will be gleaned incidentally, here and under the head of Diseases of the Arteries. Indeed, if there be no means of dealing with the local process, as by the use of potassium iodide, the management of stenosis is an eminent example of "expectant treatment."

P.S.—As these pages are being printed off, I hastily intervene to qualify or indeed to contradict my statements in the text concerning pulse delay. It seems probable that Sir William Broadbent and other authors are right after all in asserting that there may be cardio-radial delay in aortic regurgitation. The misunderstanding is one more instance of the drawbacks of making tracings without time lines. The error and its correction may be reconciled by the elevation of the whole problem into a wider generalisation. Throughout this article I have assumed that, except in advanced decay and toxic states, the systole of the heart is relatively constant in duration; this, on the researches of Cohnheim, Roy, and others, has been generally accepted. But it appears that the proposition is open to grave doubt in respect of more than one kind of heart disease. Dr. Chapman, who is good enough to keep me informed of his researches on the physiology of the circulation, sends me (1st April 1898) tracings taken from a case of pure compensated aortic regurgitation, which prove that in this case, at any rate, the systole was relatively prolonged. This observation, if well founded, will throw a new light on the failure of compensation in aortic insufficiency; it means, of course, exhaustion in a proportionately shorter period of years. The prolongation of the heart's contraction accounts for the slower transmission of the wave. The details of Dr. Chapman's case are as follows:—The pulse-rate in the tracings was (about) 75. Two tracings were taken (among others). In the first the systole occupied 0·40″ (the normal systole for this pulse-rate being 0·32″ to 0·33″). The diastole was 0·36″ to 0·39″. In the second tracing the systole occupied 0·50″ to 0·53″; the diastole 0·33″. To apply this observation, in Dr. Chapman's language, "the duration of the heart's work (on this basis) is about fifteen hours of the twenty-four, instead of ten or eleven hours." On the first tracing the cardio-radial delay was as great as 0·4″. The time measurements were made with a reed vibrating at 64 per second. If I am naturally disconcerted to find, when it is too late, that much of my text in respect of these points ought to be modified, I trust I need make no apology for our common fallibility. It would seem, from Dr. Chapman's records, that cases of aortic regurgitation differ widely among themselves.

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DISEASES OF THE MITRAL VALVE

MITRAL INSUFFICIENCY

Definition.—A diseased condition of some of the structures co the mitral valve; or a defect at the left auriculo-ventricular o venting the normal closure of the orifice during the systole o ventricle, and occasioning a backward flow of a portion of the o the left auricle.

Morbid anatomy.—(i.) *In the chronic stages of rheumatic endo* The curtains of the mitral valve are thickened and comparativ the neighbouring endocardium is also denser and more op the normal, especially in the portion extending from the great flap of the mitral valve to the base of the aortic semilun Many of the chordæ tendineæ, together with their columnæ are thickened and shortened; there are often adhesions bet curtains, the cords and the columns, as well as in some cases these and the endocardium of the wall of the ventricle. In some the chordæ tendineæ, especially the finer cords which are inse the free border of the curtain, are lengthened instead of shorten ably this is due to yielding under the pressure of the blood under surface of the mitral flap, so that the edge of the latter is into the auricle during the systole of the ventricle. Whether th be shortened or lengthened, the result is an imperfect appositi curtains at the time of ventricular contraction. The endocardiu the left auricle is also thicker than normal, especially at the ring the auriculo-ventricular aperture. From this ring extends a w milky patch of the fibrously transformed endocardium into the auri and the ventricle below. Such thickening may involve the s subjacent to the endocardium, and tend to narrow the orifice, th signs may be entirely those of mitral insufficiency and not those o tion. Duroziez (14) says, that if the orifice be large enough to a passage of the thumb the signs will be those of insufficiency, an stenosis. Much, however, depends on the condition of the internal if this be smooth, as in many cases it is, there will be signs insufficiency only; if rough, there will be those of stenosis in ad

The thickening of the endocardium is due to fibrous prolife the original inflammatory exudation, a process of development nective tissue extending into surrounding structures. Repeate of endocarditis affecting the already diseased tissue cause thickenings and retractions; the thick fibroid material compr blood-vessels, and tends to induce degeneration. Fatty deg is not often observed, but calcareous change frequently, eve case of young children. The calcified portion of the valve structure may act as a mechanical irritant producing inflammatory or necrosing changes in the tissues adjacent. A fragment of the calcareous or the necrosed material may become detached and form an embolus. Very rarely a change of the firm fibrous material into cartilage has been found (72).

(ii.) *In the chronic forms of ulcerative or septic endocarditis.*—The valve curtains, the cords and columns, or the endocardium of the ventricle may show the lesions of ulcerative or septic endocarditis, the tissues in the affected areas being destroyed by necrosis. Usually the ulcerated surfaces are covered with large vegetations. These changes in a large majority of the cases of ulcerative endocarditis—about three-fourths of the total—are found on valves previously diseased. In all such cases some of the forms of pathogenetic micro-organisms are to be discovered. It is to be borne in mind, therefore, that on the chronic morbid products at the mitral orifice a destructive disease which has no relation with rheumatism may be engrafted. In a minority of the cases the necrosing changes are slow; there is evidence to show that the process may be arrested in some areas, cicatricial tissue covering the portions showing loss of substance.

(iii.) *In rupture of the mitral valve.*—The valve curtains, cords, or columns may be ruptured. It is improbable that such an accident can occur from strain where the structures had been previously healthy. Post-mortem evidences of the rupture of a tendinous cord are not infrequent; an occurrence which has sometimes changed fairly compensated mitral inadequacy into a hopeless disablement (72). In the majority of cases I think it probable that the rupture is due to ulcerative changes. In some of them it seemed to have been due to the direct irritation of a calcareous plate or firm fibrous band operating during the movements of the ventricle; in others ulcerative endocarditis has effected the rupture. In the case of a curtain of the valve there may be first aneurysmal pouching, and secondly perforation. A vegetation on the curtain, if it induce softening of the endocardial surface, brings about a yielding under the blood-pressure within the ventricle, and a pouch is formed which projects into the left auricle; further pressure may cause rupture (perforation), when of course the valve is no longer competent.

(iv.) *In papilliform endocarditis.*—A form of chronic endocarditis is sometimes observed in which there are small, firm, warty outgrowths from the surface; these are fibrous proliferations of the endocardium, usually attached by a broad base but sometimes pedunculated. They are covered by smooth endothelium to which fibrin does not adhere; the sclerous changes of rheumatic endocarditis are not associated with them. They have been most frequently observed in cases of chorea; Lancereaux has found them also in alcoholism and in malaria. I have seen an example in a case of tuberculosis. Sometimes in newly-born infants small spherical outgrowths are observed on the free border of the mitral; they are probably hæmatomata due to rupture of blood-vessels situated under the most superficial layer of the endocardium (36, 44); usually they disappear

in the first few months of life, but in some cases they may in
warty excrescences above described (Cruveilhier). I have con
probable that in some cases of chorea, determined by sudde
similar ruptures of intra-valvular vessels with subsequent fibr
transformations occur.

(v.) *In dilatation of the left ventricle.*—There may be co
dilatation of the ventricle, and yet the mitral curtains be quite c
to close the aperture. In many cases, however, when there is
of the structures constituting the valve, the cavity is so great
that it is demonstrably impossible that the aperture between a
ventricle could be adequately closed during the ventricular systole.
the post-mortem associations of the latter condition are the f
(*a*) there may be disease at the aortic orifice causing obstr
regurgitation or, as very frequently is the case, the combined lesi
ventricle has become hypertrophied and dilated on accoun
abnormal pressure to which it has been subjected, and the
continues and progresses until the mitral curtains are no long
of closing the auriculo-ventricular orifice; (*b*) the signs of chron
of the kidneys (chronic interstitial nephritis) may be found.
cases there is great hypertrophy of the muscular wall of the
in others dilatation, even at early periods of the disease, prep
over hypertrophy. Microscopical investigation has shown
causes for the changes in the cavity and the walls of the left
are complex. The obstruction to the general arterial circulati
the thickening of the arterioles in various situations causes
intra-ventricular pressure during systole and thus there is a m
cause of dilatation; but the muscle of the ventricle also suf
the process of disease. The morbid changes in the ventric
have been described as an excessive proliferation of the c
tissue (3, 16, 29), a special quasi-inflammatory affection of th
branches of the coronary arteries — endarteritis and periarte
26, 33, 39), or a fibrosis extending to the general connective ti
starting from the arterioles and capillaries—arterio-capillary fibr
The muscular fibres are altered, the transverse striæ are obscu
fibres are atrophied and encroached upon by the fibroid tissue, c
hypertrophied. Similar changes are sometimes noted in the wa
ventricle in persons at and after middle age, when there are n
chronic Bright's disease. (*c*) As a sequence to inflammation of
cardium, the pericardial surfaces being found adherent. An
fibroid tissue not only extends amongst the muscular bundles a
but also compresses the blood-vessels; this is especially s
general rheumatic disease of the heart (carditis) in children;
ventricle may be extremely hypertrophied and dilated so that t
valve is incompetent, and yet there may be no sign of endocarditis
the structures of the valve. Dilatation of the left ventricle to t
of mitral incompetence is also observed occasionally after rheum
in childhood, with no evidence of pericarditis or endocarditis.

syphilitic affections of the ventricle the muscular fibrillæ have probably been weakened by myocarditis. In rare cases small gummata have been found in the wall of the ventricle; in others bands of fibroid material, probably the sequels of syphilitic endarteritis, and obliterations of the vessels, have been seen (*e*) in Graves' disease, and other kinds of long-continued morbid acceleration of the heart's contractions, such as tachycardia. In some such cases the left ventricle has been found so hypertrophied and dilated that the mitral curtains were incompetent; it must be remembered, however, that in many of the fatal cases of these diseases the ventricular cavity had not been dilated, and the muscle of the heart was quite normal. The dilatation of the left ventricle must be regarded only as an occasional sequel of the disturbance of the nerve-mechanism of the heart.

(vi.) *In degenerations or transformations of the structures of the left ventricle.*—In a large number of instances the various forms of degeneration of the heart—fatty, fibroid, and granular—are associated with dilatation of the left ventricle; and the mitral regurgitation, which is a feature of their history, is thus explained. In a minority there is no such dilatation. In fibroid degeneration bands of firm fibrous tissue, replacing more or less the muscular fibres, are observed, on section of the ventricular wall, to spread out in certain tracts; they often extend into the musculi papillares, and some of these may be wholly transformed into fibrous tissue. In granular degeneration, "dissociation segmenteuse" (30), the heart-muscle is observed to be disintegrated, and to present the appearance of an aggregation of fine particles; the cement substance which normally binds the fibres together being softened. This morbid condition is to be demonstrated in the musculi papillares. In fatty degeneration pale spots or streaks are observed on section, not only in the wall of the ventricle but in the papillary muscles also; then microscopic examination shows the absence, in greater or less degree, of the proper muscle elements, and the presence of minute oil globules. These transformations or degenerations may be the consequences of obliteration of the arterioles. In cases where there is sudden and recent infarction of these vessels the appearances are those of the softening known as myomalacia cordis. Where the process has been more chronic, fibrous transformations or fatty changes are observed. In some cases the two forms of transformation, fatty and fibrous, are seen together.

In another form of fatty degeneration of the heart-muscle the pale spots and the mottlings indicating the areas of metamorphosed muscular fibres are scattered throughout the ventricular wall and the fleshy columns, having no relation with any tract of vascular supply. These constitute the majority of cases known as "fatty heart." In some they are associated—especially in fat persons and drunkards—with infiltration of fatty tissue amongst the muscular bundles. Fatty degeneration of the muscular fibres of the heart is found also in chlorosis, anæmia, and blood-deteriorations. In some of these cases there has been evidence of mitral regurgitation. If there be local degeneration resulting from the obliteration of arterioles the condition leads to a fatal issue. In the majority of cases of fatty degeneration in anæmia t regains its structural integrity, and any consequent mitral in disappears. When the heart thus recovers it must be infe many of the diseased fibres actually disappear, the fat which is of their disintegration becoming absorbed, whilst new formation muscular tissue takes place. In cases in this category, where d not occur from the fatal forms of anæmia, careful search should for disease in other organs, and the formula, "death from fatty de of the heart," should not be delivered too hastily.

(vii.) In some cases in which there has been strong ev mitral regurgitation during life the heart has been found on po examination to present perfectly normal appearances. The pa such cases will be considered later.

In mitral insufficiency from all causes the *left ventricle* and its muscular walls hypertrophied. The dilatation an trophy proceed hand in hand, and both are the direct and results of the regurgitation through the mitral orifice. As th Herbert Davies pointed out, the process whereby, in sequence insufficiency, the cavity of the left ventricle becomes enlarge muscular tissue hypertrophied should not be considered mor enlargement may be in just such degree that the amount lo aorta by the leakage into the auricle is compensated; and the driving power of the ventricle is precisely regulated to d normal supply to the great artery (12).

In mitral insufficiency the *left auricle* is dilated and hype and the endocardium lining it is thicker and more opaque tha In some chronic cases the muscle of the auricle wastes, and i by fibrous tissue. The pulmonary veins also may be muc Occasionally in chronic cases globular fibrinous coagula are fou ing to the lining membrane, and projecting from between t columns and trabeculæ into the cavity of the ventricle or th These thrombi are firm and dense in their external portions, soft and fluid in their interior; cysts thus formed may rupture detached, and their fragments may cause embolism of systemi In some cases the coagula undergo fibrous and calcareous tr tions.

The *right auricle and ventricle* in cases of mitral insufficienc found dilated and hypertrophied. Hypertrophy is found to pre in the earlier stages, dilatation in the later. The wall of the v in some cases found thick and leathery, in others thin and flac tricuspid valve may be incompetent on account of extreme dil the ventricle. The globular thrombi, described as sometimes the left cavities, are much more commonly observed in the rig detached coagula cause embolisms of branches of the pulmona The dilated condition of the right chambers of the heart is associated with general venous engorgement. In the heart coronary veins are turgid and dilated.

The *pericardium* may show signs of disease, recent or remote, and there may be fluid effusion in the pericardial sac.

Morbid anatomy of other organs in mitral insufficiency.—The *lungs* in cases where there has been long-continued mitral regurgitation are found engorged with dark blood, and their fibrous tissues abnormally dense. The lung is tough; the capillaries of the alveoli have become dilated and varicose, their walls thickened. Patches showing the signs of broncho-pneumonia may be scattered throughout the toughened lung. Blood escaping into the surrounding connective tissue produces brownish pigmentation (brown induration of the lungs); it may transude into the alveoli, causing the tinged sputa and hæmoptysis observed in some cases. The lining membrane of the bronchi often shows extreme engorgement, and blood exudes from the surface. The blood-tinged sputa, therefore, may be derived from the lung capillaries or from the bronchial mucous membrane. The lower lobes, or the more dependent portions of the lung in chronic cases, become engorged, dense, and often œdematous. In many cases there are multiple pulmonary lesions, with evidence that these arose at different dates. Effusions into the pleuræ may have caused collapse of various portions of the lungs. The signs may indicate that local pulmonary infarctions have occurred in different areas at various dates. There may be the blood-clot and prominence of the pleural surface indicating a recent embolism of a branch of the pulmonary artery (pulmonary apoplexy); the sites of old infarctions may be indicated by pigmented indurations of portions of the lung-tissue, with, perhaps, some depression of the pleural surface corresponding to the indurated portion. In cases of comparatively recent embolism the corresponding area of the pleura may be covered with the yellowish exudation of pleuritis. All pulmonary apoplexies, however, are not due to infarction. The abnormal strain of the pulmonary artery may lead to degeneration of the vessel and dispose it to rupture. Old adhesions of the pleuræ or of pleura and pericardium are often observed. In many cases there is fluid effusion in the pleural cavities.

The *stomach* manifests greatly dilated veins; its mucous surface shows much congestion; the venules are often varicose; mucus, tough or fluid, is seen in abundance. The *liver* is enlarged; the intra-lobular capillaries are very greatly dilated and their walls thickened; on section it shows the characteristic appearances of "nutmeg liver," the dark brownish-red stellate spots marking the centre of each lobule on the yellowish ground formed by the bile-stained liver-cells. The bunch of greatly dilated capillaries in the centre of the lobule encroaching upon the hepatic cells may cause atrophy or fatty degeneration of the latter, some brown pigment granules being seen amongst them. The most marked signs of venous engorgement with increase of bulk of the liver are seen in cases in which tricuspid incompetence has followed mitral insufficiency. It is to be remembered that the size of the liver in such cases may become greatly reduced soon after death, the organ being partially emptied of blood by gravitation.

The *spleen* in mitral regurgitation may be enlarged fron hyperæmia, its connective tissue being much increased and caus feel much firmer than under normal conditions. In some cases infarctions old or recent. When recent, wedges of hard tissue bases at the circumference (that is, the capsule) are felt on tion. Old infarctions are indicated by shallow depressions of tl of the viscus.

The *intestines* show venous engorgement. In some cases e of the small arteries supplying the intestinal wall have been fo consequent necrosis of the bowel. The veins of the mese engorged. The glands within the abdomen are enlarged gested.

The *kidneys* are abnormally firm from cyanotic indura pyramids are especially engorged; blood may exude from the into the tubules. In some cases they show on section pal shaped, recent infarctions, their base towards the cortex and t towards the hilum; or deep depressions of the surface, with tissue visible on section, may indicate the situations of old e There may be much fibrosis in these kidneys.

The *peritoneal cavity* may be more or less filled with ascitic fl

The *membranes of the brain and spinal cord* may show muc engorgement. Signs of embolism of the cerebral arteries are some cases.

The *subcutaneous tissue* generally, especially in the lower ties, may be found infiltrated with dropsical fluid. In some case of the superficial layer of the epidermis are raised in large b other chronic cases the fibrous elements of the skin are thi there is a brawny œdema.

Mechanism of mitral regurgitation.—In normal conditio structures, after the filling of the ventricular cavity from the au muscular wall of the ventricle immediately contracts; the papillares do not begin their contraction until after an ap interval, then these muscles act with sudden energy, drawing mitral curtains and completely closing the auriculo-ventricular the apposed curtains presenting a convex surface in the auri energetic tug of the papillary muscles gradually ceases and th whilst the muscle of the ventricular wall remains contract The contraction of the muscle of the ventricle has a direct eff the auriculo-ventricular aperture. Before the beginning of th of the ventricle this orifice is circular; during the period o the contraction of the surrounding muscular fibres causes it t narrower and of oval form (35, 37). At the acme of systole the ventricular orifice has an area not much more than half that presents in diastole (35). The shape of the papillary muscle that in the complete contraction of the ventricle they are a applied to each other (63).

The ventricular systole, therefore, consists in a series of co rhythmic movements. There may be many causes of disturbance of the normal association and sequence of these actions, the result of which is insufficient closure of the mitral orifice and reflux into the left auricle occasioned by the ventricular systole. (*a*) There may be such structural disease in the curtains, cords, and attachments of the valve that due apposition is impossible. (*b*) The fibrous ring to which the flaps of the valve are attached at their circumference may be so much thickened that the muscles at the base of the heart are unable to compress it sufficiently to cause accurate closure by the curtains during ventricular systole. (*c*) The insufficient narrowing of the auriculo-ventricular aperture during systole may be due to no structural alteration of the ring, but to enfeeblement of the muscle of the ventricle. (*d*) The ventricle may be so greatly dilated, and with it the fibrous ring to which the mitral curtains are attached, that these latter fail to meet at their borders during the period of contraction of the ventricle. (*e*) The papillary muscles may be so enfeebled by disease that they fail to perform their function of approximating the valve curtains. (*f*) Owing to disturbance of the nervous mechanism the movements may not be performed in their due association and sequence.

Consequences of mitral regurgitation.—It is probable that in cases in which very small amounts of blood are regurgitated into the auricle from the left ventricle the consequences are inappreciable. The mechanical results are directly proportioned to the amount of reflux. The immediate effects may be regarded as simultaneous upon the left auricle and the left ventricle. The auricle is distended in proportion to the force of the ventricle and the amount of fluid regurgitated. The auricular wall becomes dilated, and its muscle, subjected to abnormal stimulus, hypertrophied. The left ventricle, receiving during its diastolic expansion an abnormal quantity of blood from the dilated auricle, is subjected to unusual pressure; the muscle yields and its cavity becomes enlarged. Such increase of capacity is a necessity if the normal supply to the aorta is maintained. In systole it is called upon for more work, in order to deliver an adequate amount into the aorta. Hypertrophy of its muscle ensues, and is a favourable condition. The effect of the regurgitant stream is manifested upon the right chambers of the heart. The current impelled by the right ventricle, which in normal condition should flow unimpeded through the pulmonary vessels, is met by the reflux current from the left ventricle. The capillaries of the lung, the branches and trunk of the pulmonary artery, and the right ventricle itself, are thus subjected to abnormal strain. The effects are hypertrophy of the muscle and dilatation of the cavity of the right ventricle. Hypertrophy of the right ventricle also is essentially favourable, for the more vigorous action antagonising the back flow into the left auricle helps the delivery of an adequate supply to the aorta.

The maintenance and the failure of compensation.—If the changes in the cavities and in the myocardium thus sketched out are nicely balanced, a condition of restored equilibrium ensues; thus a stationary lesion of compensated mitral regurgitation may persist for long periods, th thereof presenting neither morbid sign nor symptom. An advers however, may be effected by many causes: the dilating strain left cavities may impair the muscular power of the left au ventricle; renewed disease of the endocardium may increase tl of valvular imperfection; intercurrent diseases may affect the s integrity of the cardiac muscles, vessels, or nerves; affections of (from extrinsic causes, or from causes, such as embolism, int the cardiac imperfection) may induce direct and mechanical a indirect and enfeebling difficulties. The result of any of th ferences is a break of compensation—a failure of the cardiac circulation; the supply to the aorta and thence to the tissues inadequate, then the muscle of the left auricle and the ventricle more and more enfeebled, their constituent structures degene their cavities contain more and more residual blood. The for right ventricle now fails, and both right auricle and right become engorged with venous blood; the systemic veins are and the tissues suffer from venous stasis. The hepatic veins (v in such immediate relation with the inferior cava), being de valves, are especially congested, and their engorgement becon fest in enlargement of the liver. As the distension of t ventricle continues, the right auriculo-ventricular orifice may b much dilated that the tricuspid valve becomes incompetent to then the pulsatile action of the right ventricle is communicate valveless hepatic vein, and thus to the liver, as well as to the the neck, if the walls of these have been sufficiently dilated t their valves incompetent. The interference of the general lymphatic circulations at varying stages of this period of fail pensation may induce dropsy.

Diagnosis. — The chief sign by which the diagnosis of sufficiency of the mitral valve is to be made is a phys obtained by auscultation—a systolic murmur heard at the the heart, or having a maximum intensity in this situation. essential preliminary that the position and outline of the apex mined by palpation and percussion. The abnormal sound is some degree musical, varying in different cases from a very low high pitch; in some it may resemble the sound of a whispered " others a musical note of varying pitch and quality, and in no ir able number a shrill whistle. In many it has the sound as of steam. A characteristic to be especially noted is that it fades ally, and does not come to a sudden, abrupt stop. The murm with the systolic contraction of the ventricles; this may be de at the time that auscultation is practised, by the observer pl finger over a point where the apex beat is to be felt; or, if this be cable, over one of the carotid arteries in the neck. The bruit ma short, ceasing at an early portion of the systole, or may be p nearly throughout the whole of the systolic contraction, cea before the second sound. It may wholly replace the first sound, or the dull sound of valvular tension may be heard to precede it, when it "tails off" from the first sound. The murmur may be of very slight intensity, and may be localised at the exact apex, or it may be audible over the whole precordia with maximum intensity at the apex. In some cases it is audible from the apex in a line which extends into the left axilla, and then it often has another area of audibility at the back between the spine and the angle of the left scapula. In other cases the conduction is to the left border of the sternum above the ensiform cartilage, and the cartilages and the interspaces as far as the second left costal cartilage. I have observed cases in which the explanation of this conduction of a systolic murmur has appeared to me to be afforded by discovery at the necropsy that the disease was chiefly confined to the anterior flap of the mitral valve with the attached chordæ and the papillary muscles. Firm fibroid or calcareous material conducted the vibrations towards the septum ventriculorum, whence they were transmitted to the sternum and superficial parts adjacent. It is probable that conduction towards the axilla and the back may indicate an implication of the posterior flap in the disease.

It has been thought by some observers (41, 1, 53) that mitral regurgitation may be evidenced by a systolic murmur in the second left intercostal space, not quite close to the sternum but about two centimetres to the left of it; the murmur being due to vibrations communicated by the reflux current to the left auricular appendix (1). Many considerations seem to me to render this view untenable. The left auricular appendix, as has been pointed out by Russell, Byrom Bramwell, and others, does not approach the surface at the spot where the murmur is audible; in many necropsies it has not been visible on an anterior view of the heart; when seen it is at least an inch and a quarter to the left of the left border of the sternum, and is for the most part on the posterior aspect of the heart (55). It is more probable that the vibrations of the reflux current, if transmitted to the auricle, would be audible at the back. Duroziez has used this argument to explain the audibility of the murmur of mitral regurgitation at the back:—"L'oreillette gauche placée en arrière contre la colonne vertébrale transmet en arrière le souffle formé à la mitrale." I consider it most probable that, when the murmur of mitral regurgitation is audible in the second left interspace, it is by means of vibrations communicated to the great anterior flap of the mitral valve, or to the morbid structures in contiguity therewith.

The chief practical difficulty in the diagnosis is that of discriminating a murmur due to mitral insufficiency from one to be ascribed to the influence of the movements of the heart upon the portions of lung in front of it and around it. Cardio-pulmonary murmurs have been described by many observers (51, 64, 28, 18, 42, 48, 40). Of these Potain has made a careful and elaborate study.

The first sound of the heart to the right of the apex and over much of the area occupied by the right ventricle is often observed to be rough under conditions in which cardiac disease has no part. S sounds have been referred to many causes which it seems u to discuss. As a rule they are readily to be distinguished from due to mitral insufficiency, because they are not heard at apex nor over the situations mentioned as those to which regurgitant murmur is conducted.

In some cases, however, the difficulties are greater. In make the distinction clear, certain steps should be methodic First, *the relation of the murmur to the movements of respiration* observed. The cardio-pulmonary murmur is usually much infl the respiratory movements; for the most part it is intensified b expiration and inspiration, especially during the latter; bu becomes inaudible at the end of an expiration. If, therefore, crescendo and diminuendo in the sound of the murmur are he the respiratory acts, it is probable, though not certain, that th has its cause in the lung outside the heart.

The *position of audibility of the murmur* must be carefu Cardio-pulmonary murmurs are not heard at the exact apex ventricle, but over a sma the level of the apex to and to the left. Instead sponding exactly to the the outline of the apex ventricle, as does that insufficiency from organ these murmurs have their from a quarter of an inch and a quarter away from of apex beat. Above the there is a doubtful zone, precise diagnosis cannot made; but if a systolic m its site of maximum audi actly over the apex, it ascribed to intra-cardiac c

The rhythm of the mur be determined. A cardio-p murmur does not replace sound. The valvular flap and the murmur is observe subsequently, after an a interval, and to cease b second sound; it is manifested during a portion only of the v contraction, and is meso-systolic (Potain).

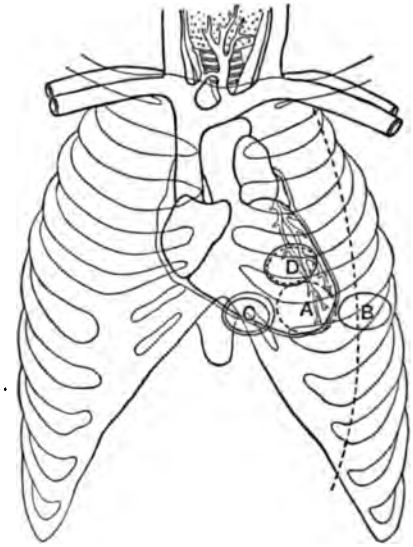

FIG. 62.—Sites of systolic murmurs at or near the apex of the heart. A, Murmur nearly always organic; B, C, murmurs always non-organic (cardio-pulmonary of Potain); D, doubtful; murmurs usually non-organic. (Potain.)

In the next place, *auscultation should be practised in various* *the patient.* A cardio-pulmonary murmur, as a general rule evident when the patient is recumbent, diminishing in inte even disappearing when the sitting or erect position is assumed. In a minority of cases this rule is reversed. It has been shown by Cuffer that though the bruits which have their causes outside the heart are in the greatest degree modified by changes of position, yet systolic apical murmurs, due to organic mitral disease, are sometimes similarly influenced. Potain says that if the change from the dorsal decubitus to the sitting position causes the complete or almost complete disappearance of the murmur, it can be confidently ascribed to extra-cardiac causes; the same may be said when a murmur well marked in the erect position disappears on recumbency. On the other hand, it is not true that every murmur which is uninfluenced by changes of position is necessarily organic.

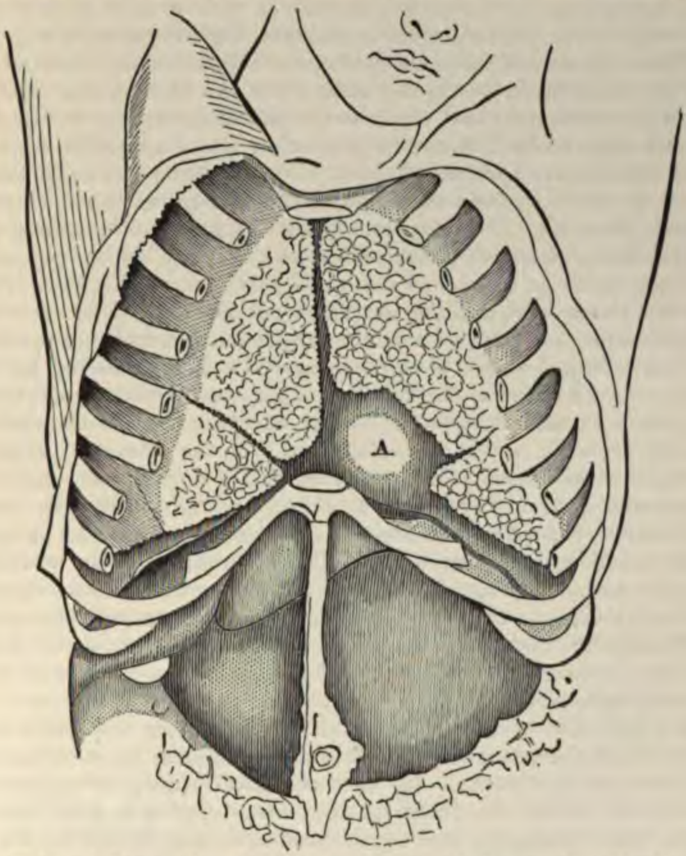

Fig. 63.—A, Portion of heart and pericardium uncovered by lungs. (After Aitken.)

Potain has adduced a great amount of evidence to show that the cardio-pulmonary murmur is caused by an aspiration of some of the alveoli of the lung produced by the cardiac movements. When the heart these are not perceptible on palpation. The line of dulness or deficient resonance on percussion, indicating the outline of the left ventricle, may be found to extend to the left of the mammillary or mid-thoracic line, even as far as the axilla at the level of the seventh rib, and thence in a line inclining upwards to the level of the second left intercostal space. The upper limit of deficient resonance has been found above the second rib (19).

At post-mortem examinations, even when there is clear evidence of much hypertrophy and dilatation of the left ventricle, the latter is generally observed only as a mere margin to the left of the right ventricle on an anterior view of the heart; the left auricle is often invisible on inspection of the front, and only discovered on so turning over the heart that a back view is obtained. It must be remembered, however, that the conditions during life differ from those observed after death; the heart-muscle contracts in rigor mortis: nevertheless, it is no doubt correct that the left auricle and left ventricle occupy but a small portion of the left border of cardiac dulness.

In cases in which a notable accentuation of the pulmonic second sound and the physical signs of enlargement of the left ventricle are manifested with no evident deviation of the right chambers from the normal, it may be inferred, that though regurgitation through the mitral orifice may be considerable, the lesion is compensated by augmented force of the right ventricle.

For the due estimation of the extent of the lesion the right cavities must be carefully explored. Palpation may detect a forcible heaving of the right ventricle to the left of the ensiform cartilage. Percussion parallel in direction with the long axis of the sternum may indicate a line of deficient resonance extending to the right of the mid-sternal line in various degrees in different cases. The dulness exceptionally extends to two and a half inches from the median line; it delimits the right border of the right auricle.

In some cases I have found on plessimetric percussion that the right border of dulness does not meet the line which indicates the upper border of the liver at a right angle; but, from one to two inches above the liver, a sloping line of dulness extends from the auricular border to meet the liver dulness an inch to an inch and a half to the right of the sternum. There is a wedge-shaped area of deficient resonance to the right of the vertical line which indicates the limit of the right auricle. I believe this to be due to a distension or dilatation of the venæ cavæ as they open into the auricle; it is only observed in cases of great dilatation of the right cavities. The upper limit of dulness may reach as high as the lower border of the second right costal cartilage. The extent of the dulness from right to left may be determined by percussion over the first part of the sternum in a horizontal direction; this line crosses the sternum to the second interspace on the left side. Such a line of dulness over the sternum at the level of the second rib still indicates the right auricle, which may even encroach on the second interspace on the left side. The remainder of the upper limit of dulness is due to the right ven the pulmonary artery.

The evidence of the outline of the heart obtained by percus not be accepted without the due estimation of causes of fall tension of the stomach with air will cause a tilting of the vent higher plane, and a dislocation towards the right of the right The content and consequent bulk of the right auricle and vent with the varying turgescence of the liver. Such distension ma tracted and due to a lasting or temporary and evanescent m gestion; for it is well known that the liver presents great var bulk even during brief periods of time. A dilatation of the blo within the abdomen (that is, in the splanchnic area) also may r content and consequent bulk of the right auricle and ventricle w is no obvious change in the volume of the liver. Another reduction in the observed size of the right cavities is expansi lungs. In such cases there are two causes of a recession of th deficient resonance indicating the bulk of the heart; namely, th air-cells of the tongues of pulmonary tissue overlapping the he give rise to a clearer note on percussion, and the augmented blood circulating in the pulmonary blood-vessels which red content of the heart chambers.

The apparent bulk of the heart, as determined by the physical diagnosis, may be temporarily increased by congesti vessels of the pericardium and neighbouring pleura as well as o the coronary blood-supply; and there may be fluid exudation spaces of the surrounding tissues.

The testimony of many observers has shown that the bul heart may be much reduced in a brief space of time—in some in acute rheumatic diseases, without relation to the therapeut adopted; in others in response to special methods of treatmen baths and certain methods of muscular exercise.

When in a case manifesting the murmur of mitral regurgita found that the right chambers are persistently dilated, and if physical signs of tricuspid incompetence be present, it must b that the degree of valvular imperfection is great and the musc heart gravely approaching failure.

The investigation of the bulk of the liver is also import guide to the estimation of the degree of valvular imperfection manifesting the murmur of mitral insufficiency. When there of dilatation of the right chambers of the heart, and the liver a thickened rounded tumour below the right costal margin, it inferred that the mitral valve is gravely incompetent—the im is still greater if the liver be felt to pulsate.

Important evidence is afforded by the observation of the of *the pulse*. If in a case manifesting the systolic apex murmur physical signs of mitral insufficiency, the hand of the observer a the precordia is sensible of a forcible ventricular contraction, radial and other arterial pulses are found to be small and weak, the inference is legitimate that much of the volume of blood which should have been delivered into the aorta is lost by regurgitation into the auricle. The pulse of a slight mitral regurgitation differs inappreciably from the normal: when the lesion is considerable the volume is small and the tension low. The sphygmograph often shows dicrotism when the evidence of impaired tension is not obvious to the finger. Not infrequently, even when compensation is maintained, the low-tension pulse presents marked fluctuation of the base line which shows that the normal correlation between circulation and respiration is disturbed.

The *cardiogram* in a case in which there is free mitral regurgitation sometimes presents special features. There is a pronounced dip or notch in the upper part of the tracing giving the summit a forked appearance.

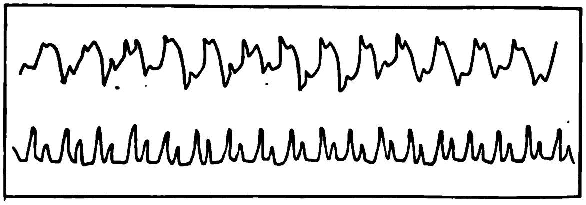

FIG. 64.—Cardiogram and sphygmogram from a case of free mitral regurgitation.

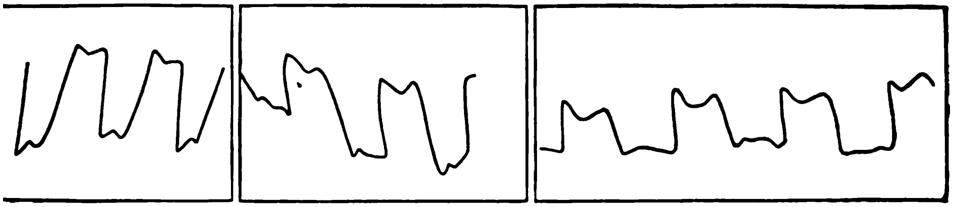

FIG. 65.—Cardiograms in mitral insufficiency showing dip or notch in the upper portion of the trace.

It would seem probable also that the relative durations of the systolic and diastolic periods, as expressed in the cardiogram, are altered; the diastolic period being relatively shortened. In compensated mitral regurgitation in many cases neither cardiogram nor sphygmogram presents any notable deviations from the normal.

Irregularity of the pulse is not, in my experience, a characteristic of mitral insufficiency.

Clinical groups of cases of mitral insufficiency.—*Group I. Mitral insufficiency the result of rheumatic endocarditis.*—It will be convenient to consider this group in two divisions: the first of children, the second of adults.

In children of twelve years of age and under, who have suffered either from a well-marked attack of rheumatic fever, or from repeated attacks motion of the heart when she strips the child to give the bath. The evidence points to the conclusion that a form of endocarditis which has the essential characters of the rheumatic may occur in infancy and childhood without any other manifestations of rheumatism (17). Endocarditis then may occur as a solitary expression of the rheumatic disease (Archibald Garrod). [*Vide* art. "Acute Rheumatism of Childhood," vol. iii. p. 42.]

The symptoms observed in childhood during the progress of uncompensated mitral inadequacy are very varied. The age of the child has some influence in regard to these. As a general rule, the signs in infants and very young children are chiefly those of inanition,—emaciation, anæmia, and deformity of the thorax. There are in many cases frequently-recurring attacks of bronchitis or broncho-pneumonia, cough being a prominent symptom. In children after the age of four years symptoms more directly indicating disorder of circulation become manifest: bleeding at the nose may be cited as one of these. Difficulty of breathing becomes apparent, and in some cases most distressing orthopnœa. Precordial pain and discomfort are severe symptoms in some cases, and these may be associated with lumbar pain. Palpitation may be a distressing symptom. Dropsy is by no means uncommon, but it rarely follows the gradually ascending course usual in the adult; the œdema is either more general, or more variable in the sites of its manifestation. In cases with œdema or ascites albuminuria is a frequent complication: this may be transient and due to venous congestion, but in the majority of cases it is dependent on the coexistence of inflammation of the kidneys, and is a sign of dangerous import. In the later stages of the disease vomiting and diarrhœa may be observed as most serious indications; hæmatemesis occurs in some cases. A marked anæmia, occasional vomiting, restlessness followed by apathy, and partial unconsciousness are symptoms which in many cases mark the weeks or days preceding the close of life.

In the form of mitral insufficiency attended with general carditis the prognosis is bad. The pericardial adhesions and the consequent hypertrophy and dilatation of the whole heart are a constant menace, and prevent satisfactory treatment. On the other hand, an uncomplicated mitral insufficiency in childhood often has a favourable issue; and the results of treatment even when the severe symptoms of threatened failure are present are often very satisfactory. Henoch considers that children recover from rheumatic endocarditis better than adults, and that in them the valve is more likely to regain its structural integrity. Cadet de Gassicourt has given his opinion that, whilst in the adult the valvular thickening increases, and the sclerosis at the auriculo-ventricular opening becomes more and more considerable, so that incurable disease remains, in the child there is more probability of absorption of the morbid products: then, the obstacle being removed, the growth of the cardiac muscle ceases to be exaggerated, and the heart, at one time too large for the child, comes by degrees into due proportion with the needs of the adult (71).

In the *treatment* of mitral insufficiency in the child when
tion fails and the symptoms are those of progressive cardiac enf
—the condition being one of chronic disease uncomplicated
rheumatism—the following are the chief points to be observed :—
in the recumbent position, or in the semi-recumbent with the
supported, must be maintained as much as possible. (ii.) Preco
and discomfort or difficulties of respiration call for the appli
warmth to the chest by warm moist flannels, spongio-pilin
jacket poultice. On some occasions a digitalis poultice may
vantage be substituted for the ordinary linseed meal poult
is made by boiling two ounces of digitalis leaves in a pint
for ten minutes, about two ounces of linseed meal being gradua
until the proper consistence for a poultice is attained. The m
course to be spread upon suitable material and applied in the u
(iii.) Means for inducing good general nutrition are of the first im
A child with mitral incompetence is often intensely anæmic.
oil, by itself or in an emulsion, or in combination with some of the
parations, is very beneficial. In some cases small doses of arsenic
solution), with tincture of nux vomica or liquor strychninæ, succe
than iron. In not a few I have seen a plan of supplementary alin
by nutritive enemas turn the scale towards amendment. One of
of such enemas is made by shaking together in a bottle one egg,
of hot milk, and an ounce of cod-liver oil, and administering ve
through a large soft rubber male catheter, with a funnel attached
at a sufficiently high level, or by an india-rubber enema tub
administration should be twice or three times daily. (iv.)
tonics are to be prescribed with judgment. In some cases rest,
regulated diet, and the tonic methods just mentioned suffice,
agents which directly influence the cardiac rhythm are unnec
even injurious. Of all cardiac tonics digitalis is of the greatest
is especially so when dyspnœa is a marked feature. The drug
given in the form of the tincture in doses of from one to five m
the infusion, ten minims to one drachm, or the leaves in pow
fourth of a grain to half a grain, repeated three times a day.
some difference of opinion whether the administration should
tinuously for long periods or in larger doses with omissions fo
days. In some instances digitalis is not well borne, and in chil
intolerance is usually shown by the occurrence of vomiting: it s
omitted whenever vomiting appears. In cases when digitalis
tered by the mouth seems to be inert, rapid improvement ma
the hypodermic injection of digitaline $\frac{1}{100}$ to $\frac{1}{80}$ of a grai
child of from six to twelve years of age. In any case such hy
injection should not be repeated for at least forty-eight hours.
alternative to any preparation of digitalis caffein citrate diss
water or in the ordinary saline mixture, in doses of from one
grains three times a day, may be given. The administration sh
be continuous, but for a period of four to six days, with simila

of suspension; for all cardiac tonics, though tending at first to increase the excretion of urine, by their prolonged action often tend to diminish it. In cases where as a consequence of mitral regurgitation the right cavities of the heart are much dilated—especially when the tricuspid valve is rendered incompetent—digitalis and other cardiac tonics may be powerless for good. Their inefficiency is readily to be explained, for it must be remembered that their action is on both ventricles, and that they augment the force of the right ventricle as well as that of the left: now increased action of the right ventricle means so much the more reflux into the general venous system and further disasters. In many cases where there is such distension of the right cavities (an occurrence which may supervene as an acute phase in a case of chronic mitral insufficiency), the relief of venous pressure by leeching is a most valuable auxiliary to treatment. One or two leeches may be applied to the precordia, and the leeching may be repeated on several occasions at intervals of two or three days. Exceptionally, half-a-dozen leeches may be applied at the first. I have often observed that digitalis, which has been powerless for good before the application of leeches, proved of great service thereafter (58).

Dropsy, in cases of chronic mitral insufficiency in the child, may be transient, and yield to the medicinal treatment already sketched out; or it may become a far more serious symptom. There may be general anasarca, and pronounced ascites and effusion may rapidly take place within the pleural cavities. In a considerable proportion of cases desquamative nephritis is manifested in the course of the mitral disease. In the treatment of such cases, sponging of the skin with hot water made alkaline with sodium carbonate, the child being afterwards wrapped in a hot blanket, is often a more practicable and efficient measure than the administration of a hot-air bath or a vapour bath. Purgatives, as compound jalap powder, are essential; at first calomel may advantageously be administered therewith. Saline diuretics are to be combined with digitalis and decoction of broom. The removal of all traces of dropsy in the child is sometimes rapid. In some cases medicinal means fail. As a rule, punctures of the skin of the lower extremities and the use of Southey's tubes in the treatment of dropsy in the child are not to be recommended; there is a danger that restless movements may cause chafing and irritation. If there be ascites, paracentesis abdominis should be performed: sometimes rapid convalescence follows this operation. Sedatives and medicines to procure sleep must be used with caution, but in many cases they are indispensable.

In mitral insufficiency, the result of rheumatic endocarditis in the adult, we find associations differing from those in the cases of children. In adult life the occurrence of general carditis and the implication of pericardium, endocardium, and myocardium in the rheumatic disease are much less common. In this sense the disease is less formidable than in the child. On the other hand, repeated storms of endocarditis in the adult increase the sclerosis at the mitral orifice and the imperfection of the curtains, cords, and columns; the thickened fibrous structures tend als gressive degrees to undergo degeneration and calcareous transf The already diseased endocardium may be attacked by pathogene organisms; the endocarditis may be septic. This is especially in women after parturition, and in both sexes when there ar of septicæmia; but the disease may arise insidiously without infection. The causes of overstrain, both physical and ment affect the adult warrior in the battle of life adversely modify ditions. Emotions disturb the rhythm of the heart and tend to compensation. Severe physical efforts may rupture curtains already diseased. Diseases of various forms may alter the nu the heart-muscle. There are probably many forms of disease aff coronary arteries and their branches within the heart; arteritis arteritis occur in many forms of infectious disease, and notably ir Arterio-sclerosis involves the coronary arterioles (especially ir Bright's disease), and the larger branches in the later periods life become affected by atheroma. The result of all such morbi tions of the walls of the arteries is an impairment of the for cardiac muscle with subsequent degenerations. Intercurrent di the lungs, again, may rudely interrupt a compensation hitherto sat In some cases causes of inflammatory irritations are imported fr out. In others infarctions or so-called pulmonary apoplexies consequences and causes of cardiac failure. Any considerable int with the function of the lungs imposes a direct obstacle to the the right ventricle. It is the energy of the right ventricle tha pelling an abnormally large volume of blood through the p vessels, and thus antagonising in the left auricle the regurgita from the left ventricle, is the effective agency of compensation.

The symptoms in the adult of a failure of the compensatory c in cases of insufficiency of the mitral valve are briefly, and in an mative way chronologically, *difficulty of breathing*, especially up but also paroxysmally; *cough*, with physical signs of œdema of of the lungs, and often of localised consolidations; and *dropsy*, extending from the more dependent portions of the body. such symptoms and from the epiphenomena of embolism and ir pulmonary and systemic, there may be recovery. When the possible restoration of the powers of compensation are reached, th is one of suffering and sadness. The recumbent position is int the lower limbs are persistently œdematous and their integumen ated; the countenance wears the hue of combined sallow lividity, the expression is one of anxiety and of a restless cra sleep, alternating with a feeble, helpless wandering of mind; abdominal discomfort from a large and tender liver; the arter becomes feebler and nearly imperceptible, and by slow degr occasional awakenings to the reality of suffering and distress, comes extinct.

In the treatment of a case of mitral insufficiency in the ad compensation is failing, rest is of the first importance. For a practitioner to prescribe digitalis or other cardiac tonics in a routine fashion for patients who manifest morbid heart symptoms is a dangerous error. Rest, careful dieting, and judicious purgation may turn the scale towards recovery, even when dropsy, and signs of much venous engorgement of the viscera, have supervened (see case by Dr. Vivian Poore, 46). In a large proportion of cases, however, the difficulties are not to be thus surmounted, and recourse must be had to drugs, whose influence is especially upon the forces of circulation; of these digitalis is the chief. Digitalis may be administered in the form of the powdered leaves, the infusion, or the tincture. One grain of the powdered leaves is equivalent to one-third of an ounce of the infusion and to eight minims of the tincture. The leaves may be administered in doses of half a grain to a grain and a half three times a day in wafer cachet or pill, alone or combined with other agents such as mercury, iron, or aloes, or other aperients. The infusion may be given in doses of a quarter of an ounce to half an ounce, or the tincture from five to thirty minims.

In many cases the daily administration of digitalis can be continued for long periods, for a considerable number of months at any rate; but great care must be taken to ascertain that the patient is perfectly tolerant of the drug, and at the outset of this treatment the effects must be noted daily: the treatment should not be continued for more than three or four days without the control of a skilled observer. Digitalis has a complex action. It has a tonic effect upon the pneumogastric nerve, whereby its power of moderating and slowing the heart's movements is increased; but further, it increases the energy of the myocardium by a direct effect upon the neuro-muscular mechanism of the heart itself. It also augments the contractility of the walls of the arteries by an influence upon the vaso-motor centres and upon the local nervous mechanism of the muscular coat of the vessels. The good effects of digitalis are manifested by its so lengthening the diastolic pause that the ventricles become more completely filled, and deliver ampler blood-waves into the general arterial system. The arteries, when moderately contracted, do not impede the blood-flow; in fact, a larger amount of blood traverses the circulation in a given time. The ventricles emptying themselves more completely, the previously dilated heart diminishes in volume. The beneficial effect of digitalis is also shown in the production of diuresis. Neither the heightened arterial pressure nor the augmented urinary outflow produced by the drug is, however, by any means constant. Variations of arterial blood-pressure under the action of digitalis have been noted by many observers to be quite independent of the slowing effect upon the heart; indeed the diuretic results are confined almost entirely to those cases that manifest œdema. It seems probable that the fluid absorbed from the lymph-spaces, drawn within the capillaries on account of the augmented rapidity of the circulation, and carried to the renal capillaries, so stimulates the kidneys as to provoke diuresis. When there is no effused lymph to be absorbed, diuresis does not result;

in fact, the urinary outflow in some cases diminishes even t and there may be hæmaturia. Digitalis is contra-indicated whe vomiting, and diarrhœa form part of the symptoms, and when t tions of the heart are rendered inordinately slow. When the ad tion of comparatively small doses is continued too long there sort of chronic poisoning; the signs are pallor of surface, coldn sometimes, attacks of faintness; it would seem that cerebral is thus produced (Duroziez, 14). I am of opinion that the da the prolonged administration of digitalis are too often ignored. effects of digitalis may persist long after cessation of the admin Abnormal retardation of the heart's contractions has been noted after omission of the drug (Raven), three weeks (Potain), 28 and (Duroziez). The practical rule should be that average doses of parations of digitalis, repeated at intervals of four hours, shoul continued, in the earlier stages of treatment, for more than thr then the drug should be suspended for a like period. It is o a patient manifests a perfect tolerance that the protracted admin should be permitted.

The employment of digitaline is preferred by many physician ally by the French. It is to be remembered that the various di vary greatly in strength; that of Nativelle has about fifteen t strength of the digitaline of Homolle, weights being equal. prescribes for a case of cardiac failure with dropsy one milligr Nativelle's crystallised digitaline. This may be administered dose, or, if tolerance be doubtful, it may be divided into fou doses given in as many days. After the administration there profuse diuresis. There should be no readministration for man the interval may be from ten days to three weeks; renewed acc of the pulse is to be taken as an indication for repetitio treatment.

Digitaline may be administered hypodermically. When sat effects have not followed administration by the mouth, I have se lent results follow the hypodermic injection of digitaline in the a solution of the discs of Savory and Moore. Each disc cont of a grain. The dose should not exceed two discs ($\frac{1}{60}$ grain).

In cases in which the right chambers of the heart are m tended the abstraction of blood is indicated. A bleeding from to the extent of six or eight ounces coincidently with the admin of digitalis, or subsequently to it, will often turn the scale recovery.

In some instances of mitral insufficiency, the consequence of r disease, the treatment by digitalis entirely fails; there seems t good effect upon the left ventricle, the right cavities continue t dropsy increases, and the drug in combination with ordinary fails to increase the outflow of urine. Other cardiac tonics and combinations of these may then be tried.

Caffein or its citrate may be given in doses of from three to fi every four hours, but, as in the case of digitalis, it is better that it should not be used for more than three days continuously. I prefer to administer it in the ordinary saline mixture (solution of ammonium acetate). Its action is in many points similar to that of digitalis, but it has a much less effect in retarding the pulse and also in causing contraction of the arteries. Its diuretic influence is decided, and, unlike digitalis, it stimulates the renal epithelium to the excretion of solids. I have found the diuretic effect to persist after the suspension of the drug. In some cases this result is coexistent with good and rapid recovery from all distressing symptoms; but diuresis may occur and persist, and yet the result be unfavourable. The drug very rarely induces insomnia; I have more frequently observed that by lessening the dyspnœa it has promoted sleep. In some subjects, however, it produces agitation, headache, vomiting, purging, and sleeplessness. The combination of digitalis and caffein may act more favourably than either drug alone (4).

Theobromine, in the form of the sodio-salicylate (diuretin), may be substituted for caffein. It is to be administered in doses of 15 grains six times in the twenty-four hours. It is freely soluble in water. It has a stronger diuretic action than caffein, and does not cause nervous agitation and sleeplessness. The diuretic effect is manifested between the second and sixth days of its administration.

Strophanthus may be administered in the place of digitalis, caffein, or theobromine. It is given in the form of tincture (two to ten minims, in chloroform water or with alcohol), or of tabellæ, each of which is equivalent to two minims of the tincture. The dose may be repeated every four hours; the same care in watching effects and suspending the administration at intervals of a few days should be used as in the case of digitalis. The action of strophanthus upon the heart by the way of the vagus and through the local neuro-muscular mechanism resembles that of digitalis (Fraser, Popper, Bucquoy); but, according to Roy and Adami, it stimulates the contraction of the papillary muscles to a far greater degree than that of the ventricular wall; these observers have shown that on repeating the dose so that the more pronounced toxic action of the drug is manifested, the papillary muscles become notably weakened, and even their power of contraction annulled. Fraser concludes that strophanthus acts upon the heart more forcibly than digitalis, but on the calibre of the arteries infinitely less. It has often a very favourable effect upon the difficulties of breathing, and, used with care, is an efficient and useful substitute for digitalis; but it is not without its dangers. Its protracted use may cause dyspepsia with diarrhœa and wasting (Lemoine), and there are some probabilities that it may lead to sudden death in the course of its administration for heart disease (Gottlieb). I cannot doubt that the protracted injudicious administration of digitalis and strophanthus —especially in those who absorb these drugs without skilled medical supervision—has often been productive of dangerous and fatal results.

The other substitutes for digitalis, *Adonis vernalis*, *Cactus grandiflorus*, etc., are not of proved importance. *Sparteine* has no notable advantage over the broom tea (decoctum scoparii) which contains it ; the useful as a diluting agent for the heart tonics already consider *vallaria majalis* will be considered in reference to the thera mitral stenosis.

Treatment of dropsy.—By the means already indicated, toge the administration of such purgatives as produce watery evac one or two purgative doses of calomel are often of service in stage of treatment—may suffice to remove all traces of drops restore compensation. In other cases where the dropsy does not the mechanical removal of the effused fluid may be necessary. by a lancet or punctures by a needle may be made into th the lower extremities, the limbs being wrapped in flannels absorbent material to take up the fluid which copiously drains the fine trochars and canulas known as Southey's tubes may be either case the skin should be previously sponged with alcohol, an antiseptic solution. The former plan is to be preferred in t a delirious or very restless patient ; the latter when the patient i enough to allow the fluid to flow gradually through the fin tubes into the receptacle underneath the bed for many ho trochar should be inserted very obliquely beneath the skin ; th of the canula should be at the extremity (and not at the sides flexible exit tube in the portions nearest the inserted canula fixed to the skin of the leg by strips of adhesive plaster ; it shou arranged so that it does not kink and obstruct the flow. It is b the anasarca is considerable, that two canulas with tubes att inserted into each lower extremity. When ascites exists, the fl the abdomen may be drawn off by the slow process of draining small canula and fine tube, or by the more rapid process of pa abdominis. I prefer the more speedy withdrawal by a com large trochar. When ascites coexists with general anasarca it a question whether draining the subcutaneous tissue or ta abdominal cavity should be first performed. When the abdom much distended the former should be practised first, for after the the intra-abdominal effusion may become absorbed. When t is considerable paracentesis abdominis should take the pr Effusions within the pleural cavity should be withdrawn at once

Agents for producing sleep or calming nervous agitation ar importance in the treatment of the failing heart of mitral ins In some cases *chloralamide* has been useful, as it is always a hypnotic. It may be given in doses of from 20 to 50 grains cachet or in weak spirituous or acidulated solutions. Each draug be made up separately. I prefer a combination of 20 or 30 chloralamid with 30 minims of dilute hydrobromic acid with of syrup of orange flowers and an ounce of pure water, ad at bedtime. Another harmless agent is *urethane* (ethyl carbama is freely soluble in water, the solution having a saline but by unpleasant taste. In doses of 15 to 20 grains at bedtime I hav induce a calm, natural sleep lasting in a case of severe cardiac failure for more than five hours, the patient being manifestly refreshed on waking. *Paraldehyde* is perhaps a little stronger as a hypnotic. It may be administered in doses of from 30 to 90 minims in diluted syrup or in almond mixture, or in capsules (each containing 40 minims); it has a powerful and unpleasant taste.

In a considerable number of cases manifesting distressful symptoms of dyspnœa and insomnia no agent succeeds so well as *morphia*. By far the best way of administering it in cases of cardiac disease is by hypodermic injection. The solution of the acetate or the hydrochlorate or the solution of morphia and atropia may be used. The first dose should be small—one-sixth or one-fourth of a grain—but this may be increased subsequently to half a grain. Care should be taken that the administration shall not become habitual.

In regard to *diet* the aliments in the condition of failing compensation in mitral insufficiency should be very simple. Milk is the best of all foods, but in some cases is hardly tolerated. In the gastric crisis accompanying the failing heart there is often a complete disinclination for food. Then peptonised milk or milk gruel may be swallowed in sipping fashion, the patient being never permitted to take a distinct meal, nor a particle of solid food (Sir Wm. Roberts). In such cases I have seen great benefit follow the administration of peptonised enemas or the cod-liver oil milk and egg enema already mentioned in the treatment of children. Brandy, if given at all, should be in teaspoonful doses with milk and wine only. Sherry, marsala, or tokay may be given in jellies. At the subsidence of the crisis, as soon as milk can be well borne, an all-milk dietary, especially if there be dropsy, should be prescribed until convalescence.

The diet and hygiene during the stage of comparative convalescence will be considered with the third group of cases.

Group II. Mitral regurgitation in chorea.—In the majority of cases of chorea a systolic murmur, having the characters which indicate regurgitation through the mitral orifice, is manifested at some period of the disease or throughout its whole course. In a large section of such cases the signs and symptoms are such as to leave no room for doubt that the imperfection of the valve has been caused by rheumatic endocarditis. In many instances of chorea there has been antecedent rheumatism; the proportion varying, according to the beliefs of individual observers, from 8 per cent (Hughes) to 30 per cent (Pye Smith), 31 per cent (A. E. Garrod), and 32 per cent (Sir Andrew Clark). There is a consensus of opinion that about one-fourth of all the subjects of chorea are or have been rheumatic. In many also of those who have personally shown no evidence of rheumatism there has been a family tendency to the disease. The doctrine has been formulated that chorea is in all instances a rheumatic affection (Roger); other observers (Stephen Mackenzie, Barlow, and Cheadle) have estimated that in from 45 to 75 per cent of the cases there are sufficient evidences of rheumatic tendency; it may be concluded, therefore, that in the majority of cases chorea is rheumatism. It must be allowed that in many of the cases th of rheumatism (reposing as it necessarily does on the stat unskilled observers, with whom as a matter of common experie every painful affection is rheumatic) can be by no means p causes of fallacy be excluded we may perhaps take it as a fai hypothesis that about half the total cases of chorea are rheu that the endocardial murmurs manifested in these patients structural disease of the valves, the result of the rheumati endocarditis. In this section of the cases the mitral incompet is the concomitant of the disease is to be estimated and trea any failure of compensation renders such treatment necessary— to the rules already laid down. The therapeutics of chorea ar elsewhere.

Nearly all observers, however, are agreed that some case are non-rheumatic. It is well known that a sudden shock or be the precursor of chorea: such a cause may operate in a case u rheumatic, but, in common with many other observers, I have cases of chorea where a sudden and violent emotion preceded in a person who showed no sign of rheumatism, nor any proclivi Dr. Stephen Mackenzie's statistics showed rheumatism and fr nearly equal, numerically, as antecedents of chorea (38). Ob generally agreed that emotional and mental disturbances have a in the immediate causation of the disease (A. E. Garrod). ' immediate cause of chorea that can be traced with any frequency usually fright, rarely mental distress" (Gowers, 20). The hear in Dr. Stephen Mackenzie's statistics of cases of chorea, was asso rheumatism in 50 per cent; whilst in 35 per cent no such asso recorded. In non-rheumatic chorea I consider that the sym signs of mitral insufficiency differ from those in the rheumatic some of these, careful examination for many days may detect n of valvular disease; then a soft and slightly pronounced systoli localised at the position of the heart's apex, may become audib is no accentuation of the pulmonic second sound; the ventric become dilated; yet the murmur, having its original characte for several years. At later periods it may become completely The late Sir Andrew Clark held that the murmurs of mitral re so frequently observed in cases of chorea disappear, in the grea of cases, within eight or nine years of the attack (10). The features greatly differ from those of mitral insufficiency due to endocarditis. The evidence of morbid anatomy completes t tion. In cases of fatal chorea wherein a soft, apical, systolic m been observed during life, the left auriculo-ventricular ori auricular aspect has been found studded and fringed with outgrowths having the signs of papilliform elevations of the en These outgrowths are firm to the touch, and are not detached l with the finger. The endocardium is smooth over them. T begin, as in rheumatic endocarditis, with a change in the epithelium and an attachment to the roughened surface of fibrous caps, but they are firm outgrowths showing fibrous hyperplasia. Their formation is not followed by the sclerous changes, the widely-spread fibrous proliferation, the retractions of valve curtains, cords, and columns so frequent in rheumatic endocarditis. On the other hand, they interfere but little with the closure of the orifice in systole, and in process of time, the endocardium remaining quite smooth, they come to have no pathological significance whatever. It seems to me probable that they may be the immediate results of a sudden overstrain and rupture of the terminal arterioles distributed to the valve structures. The immediate symptoms induced by terror or by any sudden mental shock is a blanching of the surface of the body, a contraction of the arterioles, a stimulation or over-action of the sympathetic nerve mechanism.[1] The effect on the heart at first would seem to be arrested action, afterwards palpitation. In the case of the delicate arterioles of the endocardium of the valves the result might well be ruptures ;—minute hæmorrhages, followed by thickenings analogous to those observed after the experimental production of overstrain in animals (Roy and Adami).

In cases of chorea in which there is no evidence of failure of compensation, but only a systolic murmur at the apex to indicate some incompleteness of the closure of the mitral orifice during the ventricular systole, all treatment by cardiac tonics, or by means specially directed to the valvular imperfection, is unnecessary, and probably mischievous. The therapeutic methods adopted should be those for calming the tumult of the nervous system and for ministering to a healthy nutrition.

Group III. Mitral insufficiency the result of dilatation of the left ventricle.—This group must of necessity be subdivided. In some cases the dilatation of the ventricular wall is from mechanical causes. This can be traced in the case of disease of the aortic valves, which has caused obstruction, regurgitation, or the combined lesions. For long periods no murmur is heard at the apex, but later the systolic bruit of mitral regurgitation becomes audible, and the case, which formerly presented no such signs, begins to manifest the venous congestion, the rising dropsy, and the forms of dyspnœa of mitral disease. A similar sequence may be observed in chronic Bright's disease with arterio-sclerosis. The left ventricle may for long periods show signs of hypertrophy ; then signs of dilatation are manifested more or less rapidly ; later the murmur and the signs of mitral insufficiency are observed. The ventricle has become hypertrophied, or dilated and hypertrophied, from the resistance in the aorta and the

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[1] Witness the words of the poets :—

> I could a tale unfold whose lightest word
> Would harrow up thy soul, freeze thy young blood,
> Make thy two eyes, like stars, start from their spheres,
> The knotted and combined locks to part
> And each particular hair to stand an end,
> Like quills upon the fretful porcupine.
> SHAKESPEARE.
>
> Obstupui, steteruntque comae ; vox faucibus haesit.
> VIRGIL.

peripheral vessels on account of the thickening and contract
smaller arteries. The intra-ventricular overstrain continuin
creasing — because of the augmenting arteriole-obstruction -
ventricle yields to such an extent that the mitral curtains fai
during ventricular systole.

In another set of cases there may be none of the ordinar
chronic Bright's disease, nor of thickening of the walls of th
arteries; and yet, in patients who have manifested no signs of r
nor of endocarditis, the physical signs show dilatation of the lef
and finally mitral insufficiency. In some of these it is found
that there have been arteritis and periarteritis in the vessels of
itself; in others atheroma of the coronary artery of the lef
and tracts of degeneration, molecular, fibrous or fatty, corres
the area supplied by the branches of the artery. In another
these patients being usually obese and often alcoholic, there is fat
tion amongst the cardiac muscular fibres, and the left ventr
because of the imperfection of its muscle. In yet another su
this group the heart becomes dilated to the degree of inc
of the mitral valve from a morbid affection of the nervo
Probably the nervous influences disposing to dilatation o
ventricle have been too much overlooked. I have traced a
extreme dilatation of the left ventricle coincidently with signs
of the vagus. In several cases the complete signs of dilate
and mitral insufficiency have come on in the course of Grave
these will be considered hereafter.

It is obvious from these considerations that dilatation of the lef
with mitral insufficiency, apart from structural disease of the
be the result of various and complex morbid states. It m
membered that these complex morbid conditions may coe
structural disease the result of rheumatic endocarditis, which h
been discussed.

These considerations must have their due weight in qu
treatment. In cases of arterial obstruction in the subjects
Bright's disease, and often in patients after middle life, digital
forms of cardiac tonics fail, or even do positive harm. In such
may result from the administration of arterial relaxants, and
digitalis may often be associated. Dr. Balfour considers that
cannot be safely given in cases of senile heart without a sim
unlocking of the arterioles. The cardiac tonic, therefore,
combined with iodide of potassium or sodium, or with a nitrit
nitrite of ethyl (nitrous ether), nitrite of sodium, or nitro-glyce
cases in which there is reason to suspect thickening of the wa
arteries—in the general arterial system, or in the heart itsel
course of the iodides is to be advised. Digitalis may be also ad
for periods of two or three days at long intervals. Trinitr
be prescribed if any sign of intolerance of the iodides be notic
these seem to be inefficacious, it may be administered in one-mi

of the one per cent spirituous solution; or in the form of tablets in which $\frac{1}{100}$ grain of nitro-glycerine is combined with chocolate. For continuous administration I prefer very small doses ($\frac{1}{200}$ grain) three times a day. A combination with amyl nitrite is in some cases a distinct advantage, for example, nitro-glycerine $\frac{1}{100}$ grain, amyl nitrite $\frac{1}{4}$, menthol $\frac{1}{50}$ grain, capsicum $\frac{1}{100}$ grain, with chocolate to form a tablet (Pharmacopœia of the Westminster Hospital).

When a case in this group shows signs of marked cardiac failure, such as severe dyspnœa and dropsy, complete rest in bed should be enjoined. Before the administration of any cardiac tonic it is well that purgatives be administered. A dose of calomel, three to five grains, is a good beginning; or the patient, having abstained from liquids for some hours, may take two to four drachms of sulphate of magnesia in hot water (Matthew Hay). A considerable watery discharge may rapidly reduce the œdema. The patient should be cautioned against getting out of bed, or even assuming the sitting position during the relief of the bowels, lest syncope be thus induced. The trunk should be supported by pillows and the bed-pan used.

In cases in which dropsy is not extreme, *massage* may be of great advantage. The muscles of the extremities and of the thorax should be gently kneaded. Abdominal massage should be practised with caution; to dilate the vessels within the splanchnic area may induce anæmia of the brain. Massage of the extremities aids the venous circulation, quickens the function of the absorbents, and tends to bring about a more deliberate and efficient ventricular systole.

In the grave conditions of failure of compensation it is best that the diet be exclusively milk, diluted with barley-water or peptonised. Small quantities should be swallowed at a time. Milk is a notable diuretic, and in the dropsical stages it should form the staple diet. All strong extracts of meat, which contain many products of retrograde metamorphoses, are to be forbidden; but chicken or veal broth and jellies may be permitted in some cases. In the stages of recovery three to six pints of milk may be taken in the twenty-four hours.

When the patient begins to be able to take some walking exercise, and the probability of resuming ordinary avocations comes into consideration, the question of limitation of the ingestion of fluids has to be settled. Oertel permits only 34 to 36 ounces of water, including that contained in the solid food, per diem. The best proportions of food are said to be about 1 ounce of fat, 3¼ ounces of carbohydrates, and not less than 5 ounces of proteids. A cup of tea morning and evening, about half a pint of claret, from 8½ ounces to rather more than a pint of water, and a little over 3 ounces of soup, should constitute, besides that contained in the solids, all the fluid taken during each day. The solid diet should be rich in nitrogen—for example, bread 4 to 5 ounces, meat or fish 6 to 7 ounces, with 5 ounces of chicken or game, one or two eggs, a little salad, cheese, etc., and 3½ to 7 ounces of fresh or cooked fruit (43).

As compensation is recovered, and during its maintenanc atised muscular exercise is a valuable therapeutic means. S 1854, said that "the symptoms of debility of the heart a removable by a regulated course of gymnastics, or by pedestrian even in mountainous countries such as Switzerland or the I of Scotland or Ireland" (66). This opinion sounded the note o against the routine practice of a long series of years of keeping who presented any sign of heart disease in the most complete repose attainable. Supposing that active disease be not in the cardiac tissues, a "coddling" policy, whereby the heart kept at a minimum exercise of function, is contrary to sound p and good practice. Saeterburg of Stockholm and Zander used g in the treatment of diseases of the heart, and described their ex which appeared to be very favourable in the period between 1872. The Swedish system for the promotion of good physica ment—the chief exponent of which was Professor Ling—b important agency for preventive as well as curative treatm essentials being a forced action of the voluntary muscles for give The order proposed by Ling for these exercise movements respiratory, (ii.) lower extremities, (iii.) upper extremities, (iv.) (v.) trunk, (vi.) movement of lower extremities repeated, (vii. tory movement repeated. In the Zander system mechani ances were used for the special exercising of certain g muscles. Oertel in 1884 extended the doctrine and prac advocated, in a regulated and graduated manner, the pron vigorous muscular effort in mountain-climbing. The effort o ing a hill is much more potent for good than that of walking ground. There is an increased flow of venous blood to the r of the heart; the lungs become more fully expanded, the ch the pulmonary circulation to the left auricle are more free, volume of blood delivered to the arteries by the left ventricle i The perspiration causes a reduction in the volume of the fluid b a relative augmentation of the hæmoglobin. The lympha stimulated to their task of absorption. Many cautions, howe necessary in the prosecution of this plan of treatment. If th induce unduly rapid breathing; the patient should at once co rest and make deep inspirations. It seems to me that the pla good when, with the increased muscular effort, there is no con increase of the breathing-rate—the lungs must be adequately rapidly, imperfectly, and deceptively inflated. No effort must be It is the sudden overstrain, such as occurs in running to a station, that kills. Again, great caution must be exercised in cardiac patients to considerable altitudes. Dangerous and fatal s have occurred even at moderate elevations above the sea-level.

The climbing of hills is not to every patient a possible m treatment. Systematised gymnastic exercises exclude the ne hill-climbing. The exercises recommended by Dr. Schott of are known as resistance gymnastics (Widerstandsgymnastik). The patient, loosely and lightly clothed, is instructed to breathe quietly, and to make certain movements which are gently resisted by a skilled attendant, who uses for this purpose the palms of the hands, without grasping or constricting the limbs. The movements made are (*a*) various flexions and extensions of the forearm and upper arm; (*b*) movements of the lower extremities, the patient maintaining his position by resting his hand upon a chair; (*c*) flexions, extensions, and rotations of the trunk upon the hips. A short interval is enjoined after each movement, during which the patient sits down; the exertion should be only moderate in degree, and should cause no flushing nor pallor, nor quickened breathing.

It is not possible in all cases for a patient to have the assistance of a skilled attendant, yet much good often results from a course of systematic movements executed without such aid. These should be (*a*) exercises of the arms and coincidently of the upper thorax muscles, (*b*) of the legs both in walking and with the body at rest, (*c*) flexions and extensions of the trunk; thus movements are communicated to the abdominal viscera. No heavy weights, such as clubs or dumb-bells, should be used, and the muscles of one side of the body should not be exercised disproportionately to those of the other. So far as the movements of the upper extremities are concerned, these may be accomplished by the patient, standing erect or in the sitting position with spine straightened, holding lightly in the hands a rod or cane, and lifting this by deliberately calculated actions to the fullest extent above the head the rod is then brought down behind the shoulders, the chest being thus thrown forwards. The position of the rod is to be always maintained at right angles to the spinal column; the movements are to be repeated slowly and deliberately until there is a slight sense of fatigue.

The effect of exercise of the voluntary muscles is an accumulation of blood in their vessels of supply, and a corresponding derivation from congested areas—for example, from the right chambers of the heart and engorged veins (34). "The vessels which supply the muscles of the body are capable of such extension that when fully dilated they will allow the arterial blood to pour through them alone nearly as quickly as it usually does through the vessels of the skin, intestines, and muscles together" (Lauder Brunton, 7). The conditions, however, induced by muscular exertion are very complex. There are alternate contractions and relaxations, the former compressing the blood-vessels, the latter freeing these channels; concurrently there are increased activities of the absorbents and reflex nerve-stimulations. In the movements of the trunk upon the lower extremities another set of factors comes into play. The alternate compressions and relaxations of the abdomen affect the blood-supply to the abdominal viscera. The tendency must be in the main to cause the vessels in the splanchnic area to dilate and so to co-operate with those of the muscles in relieving any turgescence of the right cavities of the heart.

The *use of baths and bathing* in the treatment of ill-compensated mitral

insufficiency can be very useful. In years past there has
doubt too great fear lest a patient presenting the signs of
gurgitation should catch cold; thus the ablutions have of
insufficient or injudicious. The use of cool or cold water
proscribed, and possibly hot baths have been too freely indulged
effect of a hot bath is evident to ordinary experience—causing
of the vessels of the skin it may induce cerebral anæmia with s
of faintness. The debilitating effect of repeated hot baths is we
On the other hand, the invigorating effect of cold tub in those
bear the shock, and of cool sponging in those who are more su
are matters of common experience. For a long period the
patients to any health resort for a course of treatment formed n
the therapeutics of heart disease. Beneke in 1859 and 1861, an
in 1878, adduced evidence to show that the baths of Nauh
Frankfort, in Germany, were beneficial in increasing the for
heart and in restoring compensation in cases of valvular disease.
Blanc in 1886 recommended the course of treatment at Aix-les
douches (temperature about 90° F.), together with skilled mass
he cited 52 cases of mitral regurgitation in which this plan was pu
15 of these all signs of disease disappeared, in 21 there was imp
and in 16 the signs remained stationary (5). The chemical co
of the water of Aix-les-Bains has probably but little to do with
peutic effect as used externally in these cases. Its chief valu
its soft, unctuous quality, due mostly to the presence of organi
(barégine), which, when at the agreeably warm temperature at
is used, adapts it admirably for the douche-massage. The th
conditions of the employment of the Nauheim waters are more
These come from hot springs (temperature 83° to 100° F.),
charged with saline matters, chiefly chlorides of sodium and calc
free carbonic acid gas. In marked feebleness of heart, and gen
the earliest stages of treatment, the patient takes a saline bath fr
the carbonic acid has been allowed to escape; the duration of th
six to eight minutes, the temperature of the water being 95°. A r
hour is enjoined after each bath. The periods of immersion are i
during the course of treatment to twenty or thirty minutes, and
perature is lowered by degrees to 85·5° F. The water used is al
retain its carbonic acid in less or greater proportion, as it is ex
longer or shorter periods to the air, or used as the Strombad foam
its full content of the gas. The effects of the various agencies th
force have been studied experimentally by Dr. R. F. C. Leith an
(31). In regard to temperature, simple thermal baths at 9
under commonly tend to reduce the pulse-rate by five or seve
minute. The effect of the addition of sodium chloride to the
generally to emphasise the change in the pulse, and to make
more agreeable to the patient; when the bath is charged with
acid gas (Sandow's effervescing tablets being used) the puls
further reduced, whilst the force of the heart's action is increa

pleasantness and buoyancy of the bath are also enhanced, and the patient experiences an agreeable sensation of warmth. The result of a bath at a temperature below body-heat is contraction of the cutaneous vessels of the area immersed, higher temperatures cause their relaxation; the lymph-circulation is necessarily modified, the internal vascular conditions are changed, dilatations of the vessels occur in various regions—notably in the vascular districts of the brain, and probably there are some rhythmic alteration of dilatations and contraction. Furthermore, there are reflex effects upon the vaso-motor and cardio-inhibitory centres. When the bath contains free carbonic acid gas the fine bubbles adhering to the skin protect the body from the colder surrounding water, and constantly impinging upon the surface stimulate the cutaneous nerve-endings. Probably also some of the gas permeates the skin; carbonic acid has been shown to be a notable and valuable local anæsthetic (Ozanam).

The effects of the combined treatment by baths and muscular exercises as carried out at Nauheim are said to be increased strength of the pulse with diminution of its abnormal frequency, decreased rate of respiration, together with fuller inspirations and greater ease and comfort in breathing, and diminution in the size of the dilated heart. There is sufficient testimony to show that in a large number of cases there has been a great improvement in the subjective conditions. The evidence is less generally conclusive as to the reduction in size of the heart. From examination of a considerable number of outlines purporting to be those of the heart before and after the Nauheim treatment, I am of like opinion with Dr. G. V. Poore, Sir William Broadbent, Dr. Leith, and Dr. Herschell, that many are the results of a fallacious plan of physical examination, and cannot be held to represent with any degree of accuracy the size and position of the heart (24, 31, 45). On the other hand, there is a very high probability that in some cases the situation and shape of the heart have become changed, and the right chambers reduced in volume. Careful observations have shown that the bulk of the heart may greatly change under varying conditions within very short periods of time. In the case of mitral disease, whilst the patient has been at rest, and when no special therapeutic means could be invoked as causes, I have observed signs of very considerable variations in the bulk of the heart in less than twenty-four hours (61). Sir W. Broadbent says: "That a diminution in the volume of the heart may take place under the influence of saline baths and certain movements there can be no doubt, but such diminution is an occurrence which is perfectly familiar to all who are in the habit of noting the changes in the size of the heart under other methods of treatment or from various causes. In a heart dilated from over-exertion, for example, the apex beat may often be felt to come in for half an inch towards the normal situation, when the patient is simply made to walk two or three times across a room" (6). Not only the positions of the apex (Leith), but also the outlines of precordial dulness, have been found to vary at intervals during the day. Heitler considers from his observations that there are rhythmic changes

in the volume of the heart, the pulse remaining unaffected by th
All these considerations must have their due weight, and to
reliance must not be placed on the evidence derived from the
means of physical examination as to the space occupied by the he
given time. The concurrence of signs,—the evidence of rational
as of physical diagnosis,—however, shows that a combination of j
bath treatment and physical exercises may be a valuable agency
in cases of mitral insufficiency with failure of compensation.

One factor in the therapeutics of a health resort must not
looked. The change in surroundings must produce an effect
higher attributes of the nervous system—the will, the emotions,
intellect. It is no slight advantage for a patient to be taken away
little worries of home to a place where, with clear sky and pure a
are facilities for systematic self-management, a prescribed and r
dietary, and the associated hope and faith inspired by the fa
experiences of others. Mental and emotional impressions can
influence the trophic nervous mechanism of the heart. It is t
there is a reverse to this picture. Patients are sometimes dec
false hopes and fallacious arguments; persons, for example, the su
mitral insufficiency, well compensated and causing no adverse sy
have been persuaded by so-called friends that calcareous incrustati
fibrous thickenings about their heart-valves would by the operat
certain "cure" disappear as crystals dissolve in water. Long and
journeys have been undertaken by those who were totally unfit
the comforts of their home, and there has followed a sad aw
from the delusive dream. These agencies are potent for good
evil, and every case in which the use of them is contemplated,
careful considered.

Group IV. Mitral insufficiency from anæmia.—A systolic
over the apex of the heart is heard not infrequently in the su
the various forms of anæmia; in some cases it is also audible at t
internally to the angle of the left scapula. Dr. A. G. Barrs f
apex systolic murmur alone in 13 out of 115 cases of anæmia.
examples of chlorosis Potain observed a murmur, which he co
to be cardio-pulmonary, in nine cases above the apex, and in one c
the apex. Byrom Bramwell and Stephen Mackenzie have record
of apex-systolic murmurs in cases of pernicious anæmia. I hav
found an apex systolic murmur in 7 per cent, and co
murmurs at the apex and over the site of the pulmonary arte
per cent of cases of anæmia. The first question to determine is
a bruit having such characters be due to causes operating ex
to the heart itself. Potain describes all the murmurs heard
neighbourhood of the heart which are causally related with anæ
chlorosis as cardio-pulmonary; he finds that they do not beg
the systolic contraction of the ventricle as organic murmurs do,
meso-systolic (occupying a portion only of the systole), that t
soft and superficial, greatly modified by the act of respiration, th

are influenced by the attitude of the patient, so that they sometimes disappear when the recumbent is changed for the erect position, and that they vary from day to day. He considers that chlorosis tends to the production of cardio-pulmonary murmurs by influencing the nervous system, and so enhancing the cardiac excitability. When in a case of anæmia a systolic murmur is heard at or near the situation of the apex, it is of importance (*a*) to determine by palpation and percussion the position of the apex beat of the outline of the left ventricle, and the relation of the observed murmur to the area thus determined; (*b*) to consider the various signs already noted which differentiate the cardio-pulmonary from the organic mitral murmur. A certain proportion may be found to answer to Potain's criteria of non-organic murmurs. I can have no doubt, however, that in some cases the apical murmur is due to veritable mitral regurgitation; first, because it has the site and characters identical with those due to organic causes, and, secondly, because it may be followed by all the symptoms of failure of compensation in mitral insufficiency. I have observed an apical systolic murmur to arise in a healthy woman after profuse uterine hæmorrhage (from fibroids), severe dyspnœa with abundant dropsy to follow, and ultimately complete recovery to take place, with the disappearance of all the physical signs of disease (56).

From the well-known association of fatty degeneration of the muscular fibrillæ of the heart with anæmia, it must be inferred that the mitral insufficiency is caused, the valvular apparatus being normal, by the resulting enfeeblement of the myocardium. The incompetence may be from impairment of the muscle of the ventricular wall or of the musculi papillares, or of both. Positive dilatation of the left ventricle has been described by some observers (Goodhart, Stephen Mackenzie, Niemeyer). In these cases the incompetency of the valve is readily explained by the passive dilatation of the auriculo-ventricular orifice; on the other hand, the ventricle, and the heart generally, have been found by other observers to be abnormally small (Duroziez, Potain). I have observed cases in which there have been the physical signs of mitral regurgitation in anæmia when the outline of the heart has been markedly smaller than the normal. The regurgitation in such cases may be explained by enfeeblement of the papillary muscles. In fatal cases of anæmia these muscles have been observed to be profoundly affected by fatty degeneration.

The treatment of cases of mitral insufficiency, the result of anæmia, is practically the treatment of the form of anæmia which is the proximate cause. Though there may be very extensive fatty degeneration of the myocardium, there is good evidence that there frequently occurs a "restitutio ad integrum"; new and healthy muscular fibrillæ being developed. The good effects of tepid and cool baths in such cases may be briefly mentioned; the use of baths, spongings, and spinal affusions of cool or even cold water has been a routine practice with many physicians, myself included, in cases of anæmia. The occurrence of a systolic murmur at the apex is no contra-indication to this mode of

treatment. The carbonic acid and saline baths, such as
Nauheim, so much extolled of late, have been used very succes
many years at Schwalbach, in co-operation with the internal ad
tion of ferruginous water, in the treatment of anæmia. The
which such baths influence the heart and blood-vessels have bee
discussed.

Group V. Mitral insufficiency in Graves' disease and allied
—Murmurs in the precordial area are heard in a large n
cases of exophthalmic goitre. In the majority of these the m
the murmurs are over the base of the heart, and especially
pulmonary artery. In a minority the systolic bruit is heard
situation of the apex. I found an apex-systolic murmur in six
series of twenty-nine cases of Graves' disease. In some of t
bruit varied much and the diagnosis of mitral insufficiency was
In one case, however, that of a lady who had previously shown n
rheumatic change in the valve, the onset and course of the dis
carefully watched, and there could be no doubt of the establis
mitral insufficiency. The disease was initiated by a sudden frig
violent palpitation the pulse-rate rose to 160 per minute, a
murmur became evident over the apex, and general dropsy superve
the usual signs of failure of compensation in mitral regurgitatio
plete recovery, however, succeeded, and the murmur disappeare
being maintained for at least thirteen years after the acute manif
(60). It is probable that in such a case the insufficiency of the v
due not to endocarditis but to a disturbance of the nerve-mech
the heart. In some cases of Graves' disease dilatation of the left
has been indicated during life and proved at the autopsy. In o
heart has been found to be quite normal. In one case of Grave
in a man, observed by myself, the dilatation was shown chiefl
right chambers; the signs of tricuspid regurgitation were mani
well-marked systolic venous pulsation in the neck. The evidenc
strongly to the conclusion that the morbid conditions of t
advanced step by step with the exophthalmic goitre, and that t
no pre-existing disease of the heart. I have found that dilatati
heart has been by no means commensurate with the rapidity of it
In cases of extreme tachycardia the outline of the heart has
normal, whilst in the case of Graves' disease in the man, w
rapidity of the heart's action was far less, there occurred distin
gressive hypertrophy and dilatation of the left ventricle. I co
probable that the insufficiency of the mitral valve, which occ
minority of cases of exophthalmic goitre—structural valvular dise
excluded—has a like pathogeny with that which obtains in anæm
valve curtains fail to coapt in some cases on account of dilatatio
ventricle; in others because of enfeeblement of the papillary mu
faulty correlation between these muscles and those of the ve
wall. In the treatment of these cases, supposing that there are
failure of compensation, the rules already laid down may be f

but another therapeutic agency demands consideration—the employment of electricity.

The treatment of the cardiac symptoms occurring in the course of exophthalmic goitre is notoriously unsatisfactory. The rapidity and irregularity of the heart's contractions in the majority, and the dilatation of the cavities in the exceptional cases, are not favourably influenced by digitalis or any form of cardiac tonic. Only those agencies which tend to calm the nervous perturbations can be relied upon. Yet I think that there is good evidence that patient and systematic electrisation, carried out in such a manner that the pneumogastric nerve and the surrounding nervous elements can be directly influenced, is of therapeutic value. The interrupted current (faradisation) as well as the continuous galvanic current were employed by the late Professor Charcot and by Vigouroux. I have not found benefit from the treatment by the interrupted current, the immediate effects of which have indeed been objected to by many nervous patients; but I consider that in the employment of the continuous current the results have been good (8). The current should be weak—two to four milliampères as given from three to eight Leclanché bichromate or chloride of silver cells: the anode should be placed at the nape of the neck, just above the vertebra prominens, and the cathode on the groove external to the larynx and trachea. The current should be allowed to pass for from six to ten minutes three times a day, the cathode, which may be moved over the skin, without lifting and re-applying, towards the clavicle, being adapted to each side of the neck alternately. This treatment in cases of Graves' disease manifesting severe and distressing cardiac symptoms has seemed to me more efficacious than any other, and a considerable number of patients have completely recovered. Although in many cases the heart-rate is often reduced after each application it is long before continuous improvement is obtained. I have seldom seen much amendment under six months of treatment.

The continuous galvanic current may also be of value as an aid to treatment in cases of failure of compensation in mitral insufficiency other than that which is manifested occasionally in Graves' disease. I have employed it in cases of chronic endocarditis undoubtedly of the rheumatic form, and it has seemed to turn the scale towards recovery. I have recorded the case of a young man who suffered from rheumatic endocarditis involving the mitral and aortic valves, and in whom extremely severe symptoms occurred during seven months. At a time when the signs were very grave the constant galvanic current from eight Leclanché cells was employed in the manner already described. Improvement soon ensued, the abnormal rapidity of the pulse was subdued, strength returned, and but for the warning note of a murmur indicating aortic regurgitation, the patient became a strong, well-nourished man (62). Potain writes concerning electrisation of the vagus: "Its efficacy is not limited to the tachycardia which accompanies exophthalmic goitre. We have been able to apply it advantageously in cases of cardio-arterial disease accompanied by marked excitability of the heart where heart remedies had absolutely

failed. It was always applied in the form of the constan
(descending), the positive pole being applied over the sides of
and the negative on the anterior surface of the chest with an
varying between 10 and 15 milliampères" (47) It is probable t
constant galvanic current we have a valuable therapeutic mea
treatment of some cases of mitral insufficiency.

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Mitral Stenosis

Definition.—A morbid condition of the structures at the left auriculo-ventricular aperture, causing a constriction of the latter and an obstruction to the normal flow of the blood from the left auricle to the left ventricle.

Morbid anatomy.—The appearances of the mitral valve and the structures adjacent to the orifice in mitral stenosis may conveniently be considered as they are manifested (*a*) in infancy and childhood, (*b*) in maturity and advanced life.

(*a*) In *infancy and childhood* the comparatively slight degrees of obstruction at the mitral orifice are marked by a ring of vegetations—in some cases friable and easily detached, in others sclerous and firmly fixed—situated around the orifice on its auricular aspect. The fibrous structures subjacent to the vegetations are firmer than the normal, the thickening frequently involving the mitral curtains, the chordæ tendineæ, and the musculi papillares. In a more advanced stage the marginal portions of the curtains are joined by fibrous adhesions. At a still later stage the two curtains are so completely fused together that the valve presents the form of a hollow cone or membranous funnel, the wider portion of which is at the auriculo-ventricular orifice, and the narrower points downwards within the ventricle near the apex of the hea funnel form of mitral stenosis, and the smooth polished membran in its conformation as a hollow cone, have suggested that the n tion of the valve is a congenital anomaly. It is undoubtedly tru rare cases such an obstruction of the mitral orifice has been found i tion with congenital malformation. In a case of this kind rec Parrot, the aorta and pulmonary artery were united in a singl In one of my own cases the aorta arose from the right ventricle, was a communication between the ventricles. In these, and in the records of which I have examined where the mitral orifice w on post-mortem examination to be obstructed in infants who die after their birth, the vegetations of endocarditis were found. I my cases, a babe of two months, a ring of granulations w encircling the mitral orifice, and the valve was thickened. I cons mitral stenosis, as observed in these cases, is not a congenital malfo but the result of intra-uterine endocarditis—the smooth and conformation of the funnel constituted by the cohering curtain valve being due to the even pressure of the fluid blood both auricular and ventricular surfaces during the rhythmic movement heart. The terminal aperture of the funnel, by which the bloo into the ventricle, may be extremely small, allowing the pa nothing thicker than a goose-quill.

The fibrous thickening of the valve, of the chordæ tendineæ may be much shortened as well as thickened—and of the papillares is in some instances very dense; in one patient, a eleven, these structures presented the characters of cartilage. Th "funnel" form of transformation of the valve is by far tl common in childhood, the "button-hole" form is sometimes obse has been noted in the case of a boy aged seven (Hayden). The ventricular orifice as seen from the auricular side then pres form of a slit or chink, or a crescentic opening in the firm, thick septum of the welded valve-structures. The division of cases o stenosis into the "funnel" and "button-hole" forms, first made b Douglas Powell, is a very practical one from the point of view of anatomy. In some cases, however, the auriculo-ventricular ape its auricular aspect presents a very irregular form. It may be sur by thickenings and nodosities, and the opening may have a p appearance resembling, as a French observer has aptly said, the anus.

(*b*) *In adults and in persons of advanced years* the "button-hole" mitral stenosis is observed with much greater frequency. In cl the proportion is about one "button-hole" to eight "funnels"; age and later life twenty-five "button-holes" to one "funnel. associations with the rheumatic form of endocarditis are abundant fested in the necropsies of cases showing constriction of the mitral adults. I have seldom, I think I may say never, observed cases of endocarditis or repeated endocarditis affecting the mitral valve—

the signs during life have indicated combined stenosis and regurgitation, or regurgitation only—without the necropsy demonstrating that the left auriculo-ventricular orifice was more or less constricted, and the surrounding fibrous ring firmer than the normal.

In many instances in adult age and later life the fibrous material is infiltrated with calcareous salts, the resulting plates having the hardness and general characters of bone. In rare cases the curtains of the valve have been found normal, whilst calcareous plates have been observed in the adjoining muscular wall of the ventricle. These may be associated with atheromatous changes, or may represent syphilitic gummata which have become calcified. In a case of chronic interstitial nephritis the vegetations surrounding a stenosed mitral orifice have been found to contain urates (Lancereaux). Dr. Goodhart, on an analysis of the post-mortem records of 192 cases, showing the changes of chronic interstitial nephritis, found that about one-fourth of the whole number presented either thickening or contraction of the mitral valve. Dr. Newton Pitt observed, on examination of the records of the post-mortem department of Guy's Hospital, that the cases of mitral stenosis in the subjects of granular kidney were to those not manifesting renal lesions in the proportion of three to one. In many cases in this category atheroma of the aorta was also found, more rarely atheroma obstructing the coronary arteries. Huchard has designated the cases as "rétrécissement mitral artério-scléreux." In some instances, as in a case of my own, chronic fibrotic changes have been found in various situations—in the pleuræ, the lungs, the capsules of the kidneys, the liver, the spleen, and the intracranial membranes. In this last, which was that of a woman aged 52, the mitral valve presented the funnel form of stenosis. This form is exceptional in the subjects of chronic renal disease, but other such instances have been recorded. It is obvious that the funnel form of transformation of the mitral valve, the so-called "pure mitral stenosis" of Duroziez and other French observers, is found not only in childhood (when it simulates a congenital malformation), but also in advanced life. In some cases it is certainly associated with rheumatism; in others such association is not proved; but it may be found in the subjects of chronic renal disease and of arterio-sclerosis.

The left auricle in cases of this affection is frequently hypertrophied and dilated. In some cases the cavity is greatly enlarged, but the walls are thin. In a child of nine years old I have found hypertrophy so far advanced that the muscle was a quarter of an inch thick (the normal being about $\frac{3}{20}$ of an inch); in another case, that of an aged woman, it was as thin as an ordinary visiting-card, almost destitute of muscle, and lined with laminated coagula. The appendix of the auricle is usually the portion which manifests hypertrophy in the greatest degree. When on opening the pericardium the heart is viewed in position, the hypertrophy of the auricle is in some cases very striking: instead of being flaccid it stands out firm and muscular. On section it does not collapse, and pronounced reticulations mark its internal surface. In other cases, when dilatation preponderates, the capacity of the auricle is increase cases enormously. The pulmonary veins are also greatly di my own records of 40 cases of mitral stenosis at all ages obs death, the left auricle was found dilated in 18, dilated and hyp in 10, and hypertrophied without notable dilatation in 3. Samways (21), who examined the register of necropsies at Guy' for four years, found that in 70 cases of mitral stenosis the l was hypertrophied in 36. In 36 cases of well-marked ste mitral orifice admitting only one finger or the extremity of a fi left article was hypertrophied in 26, dilatation coexisting in cases only was there dilatation without hypertrophy. In the ca pronounced stenosis the state of the auricle was precisely noted i and of these 5 showed dilatation without hypertrophy. The is probably correct that hypertrophy is the rule; with the hy some dilatation nearly always coexisting. When compensat the muscle becomes enfeebled, and dilatation progressively incr

The endocardium lining the auricle is usually thickened cases all over—the probable cause then being the excess of bloo to which it is subjected, and in many cases in patches by chr carditis or atheromatous change. The posterior wall of the most frequently thus affected. On the internal surface of this p auricle coagula are frequently observed. These are sometimes and composed of alternating layers of coloured and colourless fib adherent to the endocardial surface. In some cases the who thus distended with layer upon layer of coagula, resembles an (Potain and Rendu).

The vegetations observed on the lining membrane of the au be sessile or pediculated—warty, globular, or polypoid (Coa warty vegetations are simply coagula of fibrin on the diseased s the endocardium. Globular thrombi are found especially in the appendage, and between the muscular bundles; in rare cases th fill the auricle. Their external portion is smooth and tough; they are found to contain a creamy fluid. Polypoid thrombi rare; they are attached by a pedicle to the wall of the auricle auriculo-ventricular ring. Some, like the globular thrombi, a of firm fibrin; others are hard and calcified. Thrombi at auriculo-ventricular aperture are found with greater frequency stenosis than in mitral regurgitation. They may be detached an emboli, which are arrested at some point in the arterial channel or more may persistently block the aperture; or, again, one may the orifice, in the manner of a ball-valve, during certain perio cardiac cycle.

The *pulmonary veins* are in some cases much dilated; their coa thickened and atheromatous. Dr. James Barr of Liverpool has well-marked atheroma of the pulmonary veins in cases of mitral

The *left ventricle* in the majority of cases presents characters not obviously differ from the normal; its cavity is not enlarged;

instances its capacity is less than the normal. In the cases of young children the smallness of the left ventricle is striking; in some of these patients the whole heart is correspondingly diminished in size, the lungs are small, and the thoracic capacity reduced. On account of the imperfect blood-supply to the ventricle the whole organism has been impoverished (Wilks), and the entire economy has suffered from arterial starvation. In other cases the contrast with the large and muscular left auricle is very obvious. In about three-fourths of those which I observed after death the wall of the left ventricle was not hypertrophied. When hypertrophy is manifest, as in the remaining fourth of the cases, there is usually an obvious concurring cause—in the young pericardial adhesions, in the old chronic renal disease or arterio-sclerosis. Globular thrombi are sometimes found in the interstices between the musculi papillares of the left ventricle remote from the valve.

With the exception above noted, when death has occurred in the period of childhood, the *right cavities* are dilated in marked degree, and the walls of the right ventricle and right auricle are hypertrophied. The hypertrophy is often evidenced by the massive muscular columns in the ventricle and the thick interlaced muscular bands in the auricle. The orifice guarded by the tricuspid valve is usually abnormally wide; the valve in some cases is competent to close this orifice, in others its incompetence is obvious; indeed, cases have been recorded of such dilatation that auricle and ventricle appeared to form one enormous cavity.

Thrombi are observed in the right auricle and right ventricle in many cases; the surfaces of endocardium, on which they are formed, are not necessarily diseased. Such thrombi, when they become detached, plug the larger or smaller branches of the pulmonary artery. Their inception is no doubt due to the retardation of the blood-flow. The chain of consequences is as follows:—Obstruction at mitral orifice, abnormal tension of the walls of the left auricle, auricular hypertrophy and dilatation, obstruction to blood-flow from pulmonary artery to pulmonary veins, increased labour of right ventricle, tension of its walls, hypertrophy and dilatation of right cavities.

In some cases of mitral stenosis vegetations are observed on the *tricuspid valve*, and these are evidently the results of endocarditis. An induration of the structures at the right auriculo-ventricular aperture may take place, and lead to a series of morbid changes producing a stenosis of the tricuspid aperture closely resembling that of the mitral. Tricuspid stenosis is nearly invariably associated with mitral stenosis, and the morbid changes producing it are more recent in the right heart than in the left. When mitral and tricuspid stenoses coexist, the tendency to the formation of thrombi and emboli in the right cavities is more pronounced than when mitral stenosis exists alone.

In some cases the *venæ cavæ* have been found greatly dilated; the inferior in greater degree than the superior venæ cavæ.

The lungs generally present the appearances—congestions, consolidations, brown and pigmentary degenerations and scleroses—already de-

scribed in mitral insufficiency. In cases of stenosis, however, hæ
extravasations in the lungs, and infarctions of the pulmonary a
observed to a greater extent and with greater frequency. No
there are signs of pulmonary infarction, old and recent. From a
of the post-mortem appearances in 36 cases of mitral stenosis, I
infarctions of branches of the pulmonary artery, or so-called p
apoplexies, were observed in 22 instances. In rare cases a c
evidently detached from the auricle, has plugged the pulmona
itself. Cases of mitral stenosis have been recorded in which an e
dilated left auricle has compressed the left bronchus to such ext
reduce its calibre to a mere chink (Friedreich). In no incon
number of cases of mitral stenosis the lesions of tuberculosis h
found in the lungs. In the case of a woman aged 29, observed b
the necropsy showed a very narrow mitral aperture, with much t
of the adjacent structures; both lungs were studded with t
some miliary and others yellow and softening. I observed a wel
case also under the care of my late colleague Dr. Sutton. Thes
only cases of tuberculosis in association with mitral stenosis w
come under my own eye, but according to Potain the coexi
frequent. In 35 autopsies, in which mitral stenosis was demo
tuberculous changes were found in 12 instances. Taking t
recorded by Teissier, Kidd, and other observers, I find a
31 in which the association of mitral stenosis with tubercle wa
after death: of these cases 11 presented also the signs of
stenosis or of endocarditis affecting the tricuspid valve, and
manifested disease of the aortic valves. Uncomplicated mitral
therefore, was present in 16 cases only. Potain has stated his
that the occurrence of mitral stenosis in the course of pulmona
culosis is so frequent that there seems to be a causal relationship
the two diseases. Teissier has gone much farther than
considers that some form of tuberculosis is the cause, direct or he
of the "pure" form of mitral stenosis. Nevertheless, his own
tions agree with those of Letulle, that the search for bacilli an
lesion demonstrably tuberculous in the diseased structures sur
the mitral orifice has always been fruitless. To ascribe the orig
fibrous thickening to an attenuated tuberculosis seems to me
ordinary example of special pleading. A more tenable hypothes
opinion, is that in some cases the anæmia resulting from the de
an insufficient volume of blood from the imperfectly supplied
especially in the case of coexisting aortic disease, disposes to t
culous invasion; and in others the failure of the right ventric
obstruction to the supply to the pulmonary artery in the case
currence of tricuspid stenosis, disposes to tuberculosis of th
for it is to be remembered that in stenosis of the pulmonar
where there is a like physical impediment to the blood-curre
lungs, pulmonary tuberculosis is almost invariably the mode of

The stomach, liver, spleen, and other abdominal viscera stenosis show, for the most part, the appearances already described in mitral insufficiency. Embolisms and their consequences are much more frequent in mitral stenosis. Taking post-mortem evidence alone, I find that embolism is most frequently observed in the arteries of the brain and the kidneys, and these in equal proportions. Next in order of frequency are pluggings of the splenic arteries. In a small minority of cases the arteries of the pancreas, stomach, and intestines have been blocked by emboli.

In the cases in which emboli have obstructed the intra-cranial arteries the infarctions have been found almost invariably in the vessels of the left hemisphere. In seven out of eight cases the left middle cerebral artery was the vessel occluded; in two cases the anterior cerebral artery also was plugged. The resulting softening was found chiefly in the frontal and parietal convolutions and in the corpus striatum. In a case recorded by Hallopeau, in which the left vertebral artery was blocked by an embolus, softening of the left eminentia teres was observed. According to the evidence which I have obtained, fatal cerebral embolisms, which are the result of the chronic conditions of mitral stenosis, are invariably left-sided. In cases in which acute endocarditis, especially infective endocarditis, has supervened, the limitation to the arteries of the left hemisphere is not so decided. When there is necrosis of the tissues adjacent to the valve there are often multiple emboli. The clinical evidence in cases of mitral stenosis sometimes indicates a lesion of the right hemisphere, but the emboli which are fatal—probably slowly formed and comparatively large—are those which plug the arteries of the left hemisphere. There can be no doubt that the well-known physical explanation of their occurrence in the arteries of the left hemisphere is correct. The left carotid has its axial current in the same direction as that from the ascending aorta; the stream, therefore, carries the dislodged coagula most readily through the aorta into the left common carotid, the internal carotid and the middle cerebral, the current continuing in these vessels without deviation. If the embolism be large, it is sufficient to block not only the trunk of the middle cerebral artery, but also that of the anterior cerebral at its bifurcation with the former. If small, the embolism may be only in one of the branches of the middle cerebral. The right hemisphere is practically immune, because the right carotid, arising from the innominate, is placed at such an angle with the aorta as to lie off the axial current.

The working of the heart in mitral stenosis.—In the slighter forms of obstruction the mechanism is precisely that obtaining in the sclerous form of mitral insufficiency. The orifice may be so narrowed as to admit only two fingers or even the thumb only; but the thickened curtains of the valve are retracted, and the physical signs, symptoms, and consequences are those of mitral regurgitation.

The conditions are characteristically different when the mitral orifice is so narrowed or obstructed that the outflow from auricle to ventricle is seriously impeded; and when, as may be inferred with great probability, there is no regurgitation at the time of the systole of the left The most pronounced effect in such case is upon the left auri muscular wall may be greatly hypertrophied, while the diamet chamber remains not notably greater than the normal. Or, auricle may be greatly enlarged, so that in some cases its capacit than double the normal; its muscle in some cases is hyper in others atrophied, even so far as to be represented only muscular fibrillæ scattered through a shell of fibrous tissue. have differed as to the relative preponderance of hypertr dilatation in the auricle. Potain and Rendu consider that, suff immediately does a "contrecoup" on account of the obstructi the left auricle dilates and hypertrophies simultaneously, a

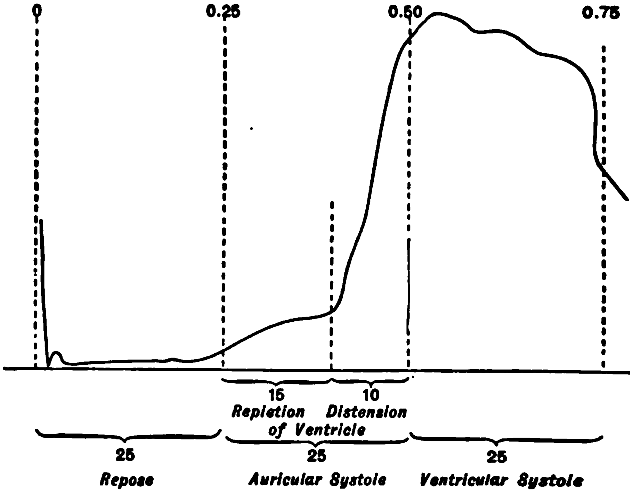

FIG. 66.—Schema of a cardiac revolution. (After Potain.)

changes are never wanting in mitral stenosis. It is obvious muscular auricle is strong enough to inject its blood-content for the ventricle even though the mitral orifice be considerably I have myself found, in the case of a child, the muscular w auricle as thick as that of the right ventricle. Cases have been in which the left auricle has maintained life for a long time whe ventricle, converted into a completely calcified chamber, incapable of any active contraction (Burns, Gérard).

It has been generally considered that the auricle ceases its traction before the systole of the ventricle begins. This was th deduced from the graphic records obtained by the experimental of Chauveau and Marey in the horse. Subsequent investigat ever, has demonstrated that the auricular systole may continue commencement of the contraction of the ventricular muscle, bo and ventricle continuing to contract simultaneously until the moment when the sigmoid valves are opened and blood begins to be expelled from the ventricle into the aorta. Potain considers that the auricle is in action from the beginning of its systole until the precise moment of closure of the auriculo-ventricular valves—that it is this muscular contraction of the auricle which ordinarily causes the propulsion of the heart's apex against the wall of the chest, and that thus it plays a notable part in the production of the impulse which is felt by the hand applied over the situation of the apex beat (Fig. 66). In stenosis of the mitral aperture this lifting of the apex by the force of the contracting auricle may be greatly exaggerated. In investigating cases of mitral stenosis by the cardiograph, I have repeatedly observed that in some the eminence which alone can be ascribed to the auricular systole has contributed to the general elevation due to the systole of the ventricle; one of the most remarkable is here figured (Fig. 67). It is to be remembered that the pen of the cardiograph is guided by the apex of the left ventricle; the record of the auricular systole is written by an impulse communicated from the auricle to the ventricle. It is obvious not only that the auricle contracts in a manner much more powerful and much more prolonged than under normal conditions, but also that it contributes in very marked degree to the general elevation which is completed by the systole of the ventricle. Professor Potain has entirely corroborated my observations and conclusions. Dr. D. W. Samways (19) has advanced the ingenious hypothesis that the abnormally powerful contraction of the left auricle prevents regurgitation in compensated mitral stenosis. He shows from mechanical and experimental data that the force of the auricle, seeing that its active contraction is continued until the aortic valves are opened and a free outflow is permitted into the aorta, is adequate to prevent any reflux during the ventricular systole. It seems to me very probable that this view is correct. It affords a good explanation of the post-mortem appearances when a contracted mitral orifice, evidently of slow pathogenesis, is accompanied by a very small left ventricle. If mitral regurgitation had occurred in such a case the ventricular cavity would in all probability have become dilated. Yet in the early stages of the transformation of the mitral orifice it would seem that such regurgitation would have been inevitable unless prevented by some cause apart from the sclerosis of the structures at the periphery of the valve. A compensatory hypertrophy of the muscular wall of the auricle—whence an abnormally prolonged

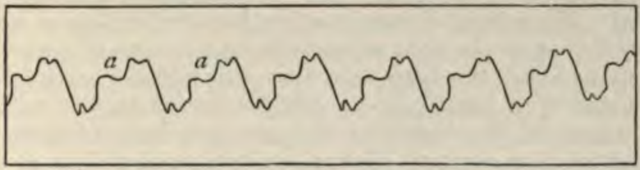

FIG. 67.—Cardiogram in a case of mitral stenosis. Auricular systole (*a*) greatly exaggerated and contributing powerfully to the elevation completed by the systole of the ventricle.

and powerful auricular systole—occurring early in the morbi
would explain not only the absence of the characteristic signs
inadequacy during life, but the absence of hypertrophy and dil
the left ventricle observed after death.

It is obvious that the enhanced force of the auricle, evidenc
muscular hypertrophy, is an important, if not the chief factor
taining compensation during the survival—many months or m
it may be—of the subjects of mitral obstruction. It is equall
that it is not the only factor, for hypertrophy of the right vent
be looked upon as a constant sequel of mitral obstruction. Dil
most cases accompanies the hypertrophy, but for long periods the
valve is competent to close the right auriculo-ventricula
Abnormal pressure is thus maintained in the pulmonary blo
The hypertrophied right ventricle co-operates with the hype
left auricle in augmenting the force by which the blood is urge
the narrowed mitral orifice. In the later stages of the affection,
the right ventricle may become dilated on account of the ex
blood-pressure to such degree that the tricuspid is no longer c
and there is reflux into the great veins. Compensation is then
maintained. The failure of compensation, however, in a given
be not by failure of the right ventricle, but on account of enf
of the left auricle. We have seen that the auricular cavity
enormous, but with practically no effective muscle in the w
evidence, especially the deposition of layer upon layer of fibr
that failure has been slow and life has been prolonged without a
participation of the auricle in the work of the circulation. T
energy of the right ventricle must have operated with the elas
of the distended auricle after its injection by the right ventricle
suction power of the left ventricle during diastole.

In a case of compensated mitral stenosis we may thus sum
work of the heart—*Systole of the ventricles.* Left unimpeded,
delivered *minus* or else sufficient for the needs of the organism
abnormally forcible, thus distending the pulmonary veins and the le
Left auricle over-distended after right ventricular systole ; this
in greater or less degree relieved immediately on diastolic relax
suction action of ventricle ; its own elastic recoil probably a
inflow into the ventricle in the earliest stages of diastole.
muscular contraction of the pulmonary veins a concurring ca
sibly such contraction in the manner of a sphincter preventi
from the auricle into the pulmonary veins ; the proper auricula
following and, being abnormally forcible and protracted, contri
produce the apex impulse.

Diagnosis.—The diagnosis is in many cases easy, in some
with considerable difficulty ; at any rate all the ordinary means o
investigation should be put in force.

Inspection may reveal no signs. The apex beat may be in
observed in the normal situation—if displaced to the left, cause

to the heart being excluded, the explanation may be enlargement of the right cavities or a general increase of bulk of the heart due, in the early periods of life, chiefly to adherent pericardium; in the later periods to the hypertrophy and dilatation of the left ventricle accompanying arterio-sclerosis. In some cases the precordial region over the right ventricle is rendered prominent and visible; pulsation is seen below the ensiform cartilage. In any case where there is this prominence over the right ventricle, whilst the left ventricle is not observed to pulsate to the left of the normal position, mitral stenosis is *prima facie* more probable than mitral insufficiency. Inspection of the veins of the neck may show a pulsation in the venous sinus just above the right clavicle coincident with the systole of the right auricle; or, when the tricuspid is incompetent, a definite pulsation of the jugular veins coexistent with ventricular systole.

Pulsation may be observed in the second interspace, or second and third left intercostal spaces near the sternum, and if a vibrating flag or lever be affixed over the spot of pulsation and another over the visible apex beat of the heart, it may be seen that the movement of the former (auricular) is distinctly in advance of that of the latter (ventricular). Thus there may be evidence of abnormal force of the left auricle. It must be remembered that this is capable of demonstration only in rare cases; it has not been observed in adults but in children only.

Palpation may reveal some very important evidence or may be negative. In a case of marked mitral stenosis of long standing a heaving impulse may be found over the position of the right ventricle, under the false ribs to the left of the ensiform cartilage, whilst there may be no palpation-signs of a forcible ventricular systole abnormally to the left. Palpation may thus confirm inspection in indicating that the right side of the heart is enlarged and the right ventricle hypertrophied, whilst the left ventricle does not show these abnormalities. Any such deduction, however, must be made cautiously, for the left heart may be more enlarged than the signs indicate, as it may be covered by inflated lung-tissue. There is one sign obtained by palpation to be observed in a considerable number of cases of mitral stenosis which, provided it has certain essential characters, may be regarded as almost a crucial sign of the affection. This is *thrill*—"frémissement cataire." The feeling of vibration communicated to the finger lightly laid in the intercostal space close to the point of the apex beat or slightly to the right thereof may be fine or coarse, protracted throughout the whole diastole and ceasing (usually) at the instant of the shock of the apex beat, but sometimes very shortly after the commencement of this event, or occupying a very brief period just before the systole of the ventricle. It can best be timed by the finger of the observer's free hand placed over the carotid artery, when the thrill is found to cease at the moment of the carotid pulse. If in the case investigated there be well-marked signs of incompetency of the aortic valves, it is to be borne in mind that the diastolic-presystolic thrill may be present without mitral stenosis. Such cases are, however, rare; in a larger number mitral stenosis coexists with the disease of the aortic valves.

In the absence of signs of aortic-valve disease a well-marked presystolic or presystolic thrill when observed in the apex region always indicative of mitral stenosis. It is important that thrill b gated in varying positions of the patient. Vibrations which are felt when the patient is in the recumbent position may beco more marked in the sitting posture with the body bent forwar observation, however, does not excuse the omission of all other means of investigation. It is to be remembered that thrill may l at some periods and present at others during the observation o Sometimes it is absent when the patient is at rest, and develo exertion or when the arms are elevated. *Percussion* is chiefly o ance to determine the outline of the heart: it gives more pre the evidence obtained by inspection and palpation, and when d tionate enlargement of the right chambers of the heart is thus i this method of investigation is valuable for diagnosis not onl nature of the affection, but of its extent and significance. T obtained by *auscultation* are of chief importance in the dia mitral stenosis—they are murmurs, double shock-sound during t of ventricular diastole (reduplication of the second sound), acc of the pulmonic second sound, loud and sudden snap at the acme tricular systole, and inaudibility of the second sound at the hea The murmur characteristic of mitral stenosis is that known as systolic murmur. It is generally of rough quality, vibratory or l Potain states that it rarely has the characters of a blowing sound most frequently it is snoring or rolling. It may begin almost imn after the second sound of the heart, be prolonged through t period of ventricular diastole, become reinforced towards the en period in a "crescendo" manner, and end with a sudden tap This terminal tension sound is in some cases coincident with the of the apex as felt by the finger; in others it is noted to o shortly after the first shock of the impulse; but it is always syn with the pulse felt in the carotid artery. The sound of the may begin long before the proper systole of the auricles (see —it may therefore be correctly designated diastolic-presystol evidence leaves no room for doubt that the reinforcement tow close is coincident with and due to the muscular contraction of th Sir Wm. Gairdner uses the term "auricular systolic" (A. S. mu denote the bruit. Whether the term "presystolic" or "auriculo- be used, it must be remembered that the active muscular contr the auricle is not the only force on which the murmur depends. cases the bruit is not prolonged throughout the periods of dia presystole (the entire diastolic murmur of Bristowe), but is aud short murmur closely following the second sound (early diastolic r or isolated with a pause before or after (mid-diastolic or meso murmur). Usually these disjointed murmurs are found in a case some periods of observation manifests the more typical presystolic

The sudden snap which generally terminates the murmur is and characteristic. In some cases it is observed without any bruit leading up to it. It is evidently an unusually short and sudden first sound of the heart; if in any case it be observed in the near neighbourhood of the apex—in some cases I have noted it at the back under the angle of the left scapula—mitral stenosis should be suspected and the concurrent signs searched for. The cause of this phenomenon is not definitely settled. It closely resembles the sound of sudden tension which may be imitated by abruptly stretching a piece of moist membrane. In the left ventricle of some hearts with stenosed mitral aperture observed after death, in which the phenomenon had been manifested during life, it would seem that there are no structures likely to give rise to this sudden sound of tension at the moment of contraction of the ventricle—the mitral curtains being thick and leathery, the chordæ shortened, and, with the papillary muscles, forming thick fibroid bands; the muscle of the ventricle not obviously differing from the normal, and the ventricular cavity small rather than large. On the other hand, the tricuspid valve is seen to be thin and membranous, and it seems probable that to its sudden tension by a forcible right ventricle the loud snap may be ascribed.

Another very important auscultatory sign for diagnosis is the double sound heard during the period of the diastole of the ventricles. This phenomenon, which vividly recalls the "postman's knock," has been generally named the *reduplicated second sound*. To avoid speculation as to its mode of production we may be permitted to call it a double-shock sound in diastole. It may be manifested in the neighbourhood of the apex or at the base of the heart. When audible in the neighbourhood of the apex of the heart and not over the base the double-shock sound indicates an early stage of mitral stenosis. This view which I enunciated in 1880 was confirmed by Cheadle. As a sign of mitral stenosis in later as well as earlier stages, it has been noted by many observers (Potain, Rouchès, Gérard, Phear, Boyd). The explanation of the mechanism of this sound first recorded by myself has been for the most part accepted. It is not a true doubling of the second sound, and cannot be ascribed to the asynchronous closure of the semilunar valves of the aorta and the pulmonary artery, but is of mitral origin. It is a sound of tension due to the first inrush of blood into the ventricle, such inrush being more sudden and forcible than under normal conditions from the increased blood-pressure in the left auricle due to the constriction of the mitral orifice. Potain, Rouchès, and other French observers have described this sound as the "claquement d'ouverture de la mitrale." Potain thus explains the mechanism of the sound. The opening of the mitral valve is normally noiseless; but in the subject of mitral stenosis the valve curtains at the moment when they separate, moved by the blood-wave that enters the ventricle, are abruptly checked by the adhesions of their free borders; the sudden tension which results produces the sound, which is the more dull as the normally thin curtains have become more dense and have lost their elasticity.

When the double-shock sound is audible over the base of the heart and not in the close neighbourhood of the apex the problem of by no means admits of a ready answer. It is undoubtedly over of the heart that the double sound, when manifested in mitral is heard in the majority of cases. The diagnostic value of th very great; the double sound either at base or apex is foun than one-third of all cases of mitral stenosis. The generally view of its mode of production is that the semilunar valves of and the pulmonary artery respectively do not close in normal syn but those of one vessel coapt in advance of those in the other to the relative degrees of blood-pressure. The objection to this h I take to be that it involves an admission of an asynchronous the two ventricles which physiologists are not able to accept. T of tension of the aortic valves cannot be produced until the left begins its diastolic expansion; if this sound be followed by th tension of the sigmoid valves of the pulmonary artery, it follows diastolic expansion of the right ventricle is not synchronous wi the left, but is in all cardiac revolutions delayed. Potain has, more recently minimised or overcome this difficulty by adva following hypothesis: Premising that the precession of the two tension is aortic in the earlier, and pulmonic in the later phas disease, he considers it probable that when the obstruction at t orifice is slight, but yet sufficient to bring about some difficu entry of blood into the left ventricle, the aspirating power of th diastole is augmented ("elle est moins aisément satisfaite"), and lunar valves, drawn upon with more force than ordinarily, c rapidly. Later, when the obstacle at the left auriculo-ventricu has notably impeded the circulation in the lung, and the right has become hypertrophied, the over-pressure in the pulmona compels the semilunar valves of this vessel to close more fo more rapidly at the beginning of ventricular diastole. For my though there is room for much difference of opinion, I think probable that the phenomenon has a similar cause at base an the heart. The first element of the double shock-sound is t second sound often accentuated as to its pulmonary artery co the second element is a sound of tension produced by the forc of blood into the ventricle, the shock being communicated eit wall of the ventricle or to the anterior curtain of the mitral v to the aortic cusps, and thence to the sternum and especiall border.

Accentuation of the pulmonic second sound is a sign to be noted stenosis as in mitral regurgitation. The cause—over-pressu pulmonary artery—occurs in both morbid states, though from causes. In mitral stenosis, however, the irregular rhythm of in many of the cases prevents a due appreciation of this accentu sound then is very loud in some cardiac cycles, in others feeble inaudible.

Another auscultatory sign to be noted in a section of th *inaudibility of the second sound of the heart at the apex.* This extinction of the second sound at the apex is usually manifested in the later stages of mitral stenosis (Broadbent, Acland); its causes are—(*a*) a diminution of blood-supply to the aorta, and consequent feeble recoil against closed aortic valves (it is the aortic element of the second sound that is audible over the heart's apex); (*b*) the enlargement of the right auricle and ventricle which, coming more and more to the front, displace the left ventricle, the chief conductor of the sound.

In the latest stage of mitral stenosis the presystolic murmur may be inaudible, the second sound absent, and the short and sudden first sound, to which attention has been already called, the only notable auscultatory sign. More frequently, however, in later as well as in earlier stages, a systolic murmur is to be heard in the neighbourhood of the apex. This murmur may have the ordinary characters of that of mitral insufficiency, audible over the apex and at the back under the angle of the left scapula, or may be a short systolic "puff" having a very limited area of audibility, but over the site of the apex. It may coexist with the presystolic murmur, which in such case is usually heard for the most part slightly to the right of it; or it may be heard when no presystolic or diastolic-presystolic bruit is audible. Nearly always in these cases the sudden tap indicating the first sound is heard over some part of the apex region. In another section of the cases the systolic murmur has an area of audibility to the right of the apex, encroaching more and more on the tricuspid region, and in some instances localised at the base of the ensiform cartilage, that is, the area of a tricuspid regurgitant murmur. It has been considered (and the contention has a great show of validity) that in some of the cases in which a systolic murmur has been ascribed to regurgitation through the mitral orifice the real cause of the phenomena has been tricuspid reflux (Samways, *loc. cit.* pp. 64 *et seq.*). In some instances, however, there are two areas of audibility of the systolic murmurs, when it is most probable that there is regurgitation through both mitral and tricuspid orifices. If the hypothesis be correct, that the abnormally powerful muscular contraction of the left auricle prevents regurgitation in the compensated stages of mitral stenosis, it is probable that some such regurgitation is inevitable when compensation fails and the auricular muscle has become feeble.

It is to be noted that a very marked irregularity of the heart's rhythm is by no means infrequent in mitral stenosis, and that this irregularity may modify all the physical signs already described. The murmurs, the so-called reduplications of normal sounds, the snap sound, and the thrill may be observed in some cardiac cycles, and may be absent in others. The irregularity of rhythm is evident to the auscultator. Such irregularity may be entirely due to disturbances of the nervous mechanism, and may be quite independent of structural changes in the heart; but when signs of organic valve disease coexist with it, mitral stenosis is the lesion in the great majority of cases.

Cardiographic evidence.—The use of the cardiograph has instances afforded valuable evidence not only for the dia the condition of mitral stenosis, but for the elucidation of so difficult problems connected therewith. The chief signs rec the cardiograph have been—(*a*) An abnormal magnitude of tion denoting the auricular systole. It has been shown in s that the power of the auricle is sufficient to lift the ventricle

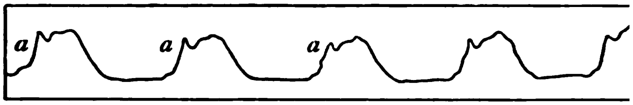

Fig. 68.—Cardiogram in a case of hypertrophy of the left ventricle. Auricular sy contributing very slightly to the elevation completed by the ventricular sys

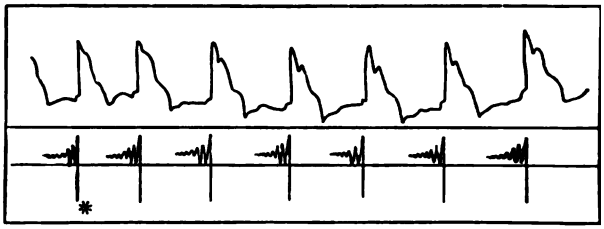

Fig. 69.—Cardiogram in a case of mitral stenosis with presystolic murmur and thrill. T shows the auricular systole in the normal position. The lower line indicates the p cardiac cycle of the vibratory murmur and presystolic reinforcement. * First soun

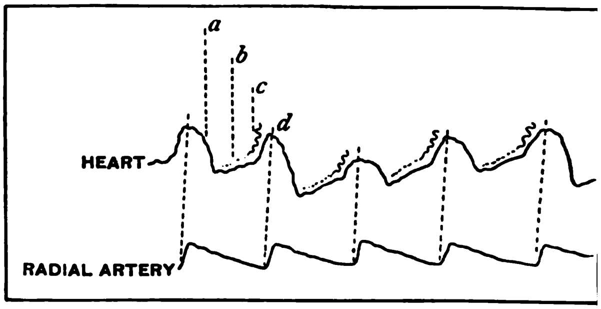

Fig. 70.—Trace in mitral stenosis. *a*, Second sound; *b*, diastolic roll; *c*, presystolic re *d*, first sound. (Potain.)

nounced manner (Fig. 66). This can only occur when the of the mitral orifice is not considerable, the auricle being hype (*b*) An increase in breadth of the auricular eminence, the summi is seen to be broken by undulations, a condition felt by the thrill. (*c*) Repeated elevations denoting rise and fall of press ventricular diastole, not necessarily indicating any muscular co of the auricle, but probably expressing graphically the interr the flow of blood through the diseased valve-structures which a as a rolling or bubbling murmur. (*d*) Fine serrations in the di presystolic periods audible as harsh murmur, and due to the causes already considered. This form of cardiogram denotes a considerable degree of stenosis contrasting with (*a*), in which the orifice is only moderately constricted. It has already been noted that in some cases (*e*) the cardiogram in mitral stenosis differs in no appreciable way from the normal. It would be legitimate in such case to infer that the stenosis is slight in degree.

Sphygmographic evidence.—In a large number of cases of mitral stenosis the sphygmograph indicates a very notable irregularity; this irregularity may be observed when the lesion is compensated and the patient appears to be in perfect health. In some instances in which the rhythm of the heart is apparently regular, a slight exertion serves to provoke the irregularity. The administration of digitalis may produce or increase it; but it is often found in cases in which the drug has not been administered. The most frequently observed form of irregularity is that evidenced in the sphygmogram by a repeated elevation in the down-stroke. There may be two or three of such elevations before the base-line is reached. It is evident that these excursions from the down-stroke contain all the elements of a complete pulsation effected by the ventricle. They show that after a comparatively effective emptying of the ventricle there may be repeated systoles following at very brief intervals. In late

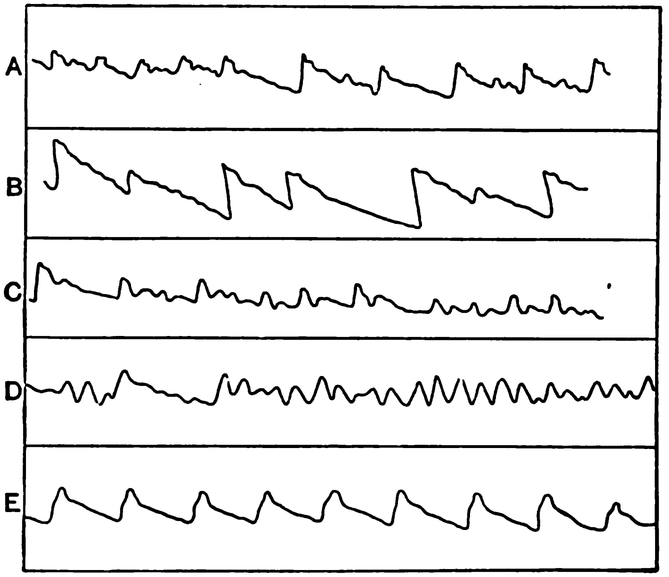

FIG. 71.—Sphygmograms in mitral stenosis. A, In stage of compensation; man aged 44, observed during five years. B, Case manifesting typical presystolic murmur; no signs of failing compensation; patient in good health. C, Mitral stenosis with failure of compensation (tricuspid regurgitation, pulsating liver). D, Late stage of extreme mitral stenosis (female aged 17). E, Regular anacrotic pulse; mitral stenosis in a female aged 41, with rheumatic antecedents.

stages of the disease the irregularity may be extreme. The pulse of mitral stenosis has been noted by many observers (Ba Mahomed, Foster, and others). I consider that the sphyg indication of irregularity in a case in which compensation app perfect may aid in the differentiation between mitral stenosis a insufficiency, for the latter lesion during the stages of compe not attended by irregularity of the pulse unless there be some neurosis.

In another large series of cases the sphygmograms show regularity in the heart's rhythm. Many observers have consid regularity to be the rule in mitral stenosis (Hayden, Fagge, Br The up-stroke of the tracing indicating the volume of the inconsiderable, and the indications are that the vessel is full be beats. Sir W. Broadbent (5) considers that this modified hig pulse is almost constant in mitral stenosis, and indicates resista capillaries. Such resistance may be due to contraction of the consequent upon the overloading of the blood with impuriti from defective elimination or, possibly, from the backward press veins effected through the capillary network, or from the cont the entire arterial system upon a diminished supply of blood imperfectly filled left ventricle.

Practically the observation of a heaving impulse of the right without signs of dilated left ventricle, together with the evid pulse having the characters above stated, may have a valuable the diagnosis of mitral stenosis. That the arteriole resistance i cases increased is proved by the anacrotic form of pulse whic times observed (see Fig. 71, E). The association with arteri well proved in a section of the cases, is in these a sufficient expl

Some difficulties in the diagnosis.—Although the presystolic and the thrill observed in the positions mentioned close to t apex are indications of mitral stenosis, in the great majority of are not absolutely pathognomonic.

Austin Flint was the first observer to show that a murmur h characters of that of mitral obstruction could be produced in insufficiency of the aortic valves in the absence of mitral stenosi observations were confirmed by many observers. I have show presystolic thrill of mitral stenosis also can be exactly simula conditions of aortic regurgitation. Dr. Phear (15) has carefully the records of forty-six cases in which there was presystolic ape without mitral stenosis; in twelve of these, thrill, presystolic or was present. In seventeen of the cases the aortic valves were inc in twenty the pericardium was adherent; in the remainder the valve-lesion, but in some of these there was dilatation of the left The hypotheses which have been adduced to explain these phen the following: (i.) That in the cases of aortic regurgitation the re stream tends to lift the great anterior mitral curtain, and so t the mitral orifice at the end of diastole as to impede the current auricle; (ii.) That the mitral valve is thrown into vibration by the two currents, the regurgitant from the aorta and the direct from the auricle, such vibrations lasting until the commencement of ventricular systole; (iii.) That in the absence of aortic valve disease, but in the presence of adherent pericardium, vibrations may be set up by the current propelled from a dilated and hypertrophied auricle into a ventricle whose muscular walls are deficient in their normal nerve-tone; (iv.) That shortening of the chordæ tendineæ, or dilatation of the left ventricle, may bring about a virtual narrowing of the aperture through which the blood passes from auricle to ventricle, the auricular muscle continuing to be sufficiently powerful to generate a fluid vein. It must be admitted that these opinions are for the most part conjectural, but the fact remains that in some cases the physical signs have led most competent and careful observers to an erroneous diagnosis of mitral stenosis. The practical lessons I take to be the following:—In cases where the concurrent signs indicate dilatation of the left ventricle, and where the previous history tells of an antecedent pericarditis, we must be cautious in interpreting a presystolic murmur as pathognomonic of a stenosed mitral orifice. In all cases careful investigation must be made into concurring signs of incompetency of the aortic valves. If the murmur of aortic regurgitation be absent from the base of the heart and the line of the sternum, it may yet be found alone at the apex, and may then closely simulate the murmur of mitral stenosis. In such case, however, according to my experience, the terminal tension sound, the tap or snap, is not marked—the sound is dull. All available means, including the use of the cardiograph and sphygmograph, should be used to effect the differentiation.

It must be remembered that aortic insufficiency and mitral stenosis may coexist, and the diagnosis of the combined lesion may present great difficulty. Dr. F. J. Smith found on examination of the post-mortem records of the London Hospital evidence of the combined lesions in thirty-nine instances. Uncomplicated aortic insufficiency was to aortic insufficiency plus stenosis as 88 to 39. The association of the two valvular affections therefore is not very rare, and the diagnosis of such association can only be made with an approach to certainty when there are decided physical indications of each separate morbid condition.

CLINICAL GROUPS OF CASES OF MITRAL STENOSIS, THEIR SYMPTOMS AND TREATMENT.—**Group I. Cases associated with rheumatism.**—The intimate relation between mitral stenosis and rheumatism is shown by a large series of cases. In some of these the rise and progress of the endocarditis, the cause of the obstructive lesion, can be traced by clinical observation. The patient may show all the signs of acute rheumatism, an occurrence comparatively rare in children, the acute symptoms being often very slightly pronounced, though in some instances they are fully manifested, and then usually the first sign of implication of the valves is the systolic murmur of mitral regurgitation. The child in the course of months or years may suffer from repeated attacks of acute rheumatism, and after

a longer or shorter interval the systolic murmur is prece
presystolic murmur, the other signs of mitral stenosis concur
some such cases, and in course of time, the murmur of
gurgitation becomes replaced by that of mitral stenosis.
instances have come under my observation (27). In other
presystolic murmur of mitral stenosis after repeated attacks of r
has been very decidedly modified—it has been followed by
murmur. The significance of such a change it may be
some cases to estimate. The murmur may be very loud, and
the left axilla and at the back: if so, there can be no doubt tha
to regurgitation from organic disease. Or it may be hea
very restricted area, not conducted to the axilla, but just
apex itself. In such case the auriculo-ventricular orifice m
widened by any retraction of curtains or columns, but the
lesion may be stenosis nevertheless, and the auricular muscle
come weak; therefore regurgitation, which previously had been
is now permitted. Or the murmur may be observed to the ri
position of the apex close to the tricuspid area; in such
probability of tricuspid regurgitation must be borne in mind.

In some cases rheumatic phenomena are declared, not in
stages of the affection, but subsequently, during the observat
case. For instance, a girl of fourteen, without any rheumatic
—though there was hereditary tendency thereto on the moth
manifested a prolonged systolic and a short presystolic mitra
There were no rheumatic phenomena for thirteen months
articular rheumatism appeared. At that time a marked thrill
the apex; a grating presystolic and a prolonged blowing systol
were heard, and the heart was enlarged, especially as regards
chambers. The autopsy showed a funnel-shaped transformat
mitral valve and a ring of small vegetations (recent rheum
carditis) encircling the auriculo-ventricular orifices. This a
of many pieces of evidence that the rheumatism which is
with mitral stenosis may be attended for long periods by no obv
toms.

A sign of the advent of the structural change in
inducing obstruction at the mitral orifice is a double sh
heard during the period of ventricular diastole, and res
doubling of the second sound over the apex of the heart. I
simulated doubling of the second sound at the apex in a large
cases which eventually manifested all the usual signs of the le
Cheadle found "33 cases with presystolic murmur, and 24
duplicated second sound at the apex, indicating commencing st
of 273 cases of organic heart disease in children" (8).
"There can be no question as to the connection of this mor
with early mitral stenosis, and of its clinical significance."
confirmed these observations, ascribing it, as I do, to causes af
mitral valve. The first element is the normal second sound h

apex, the second element occurring soon after it, the "claquement de l'ouverture de la mitrale."

In a large number of cases the clinical signs of association with rheumatism are insignificant. The evidence of many which I have analysed shows that in a considerable proportion the origin and progress of the morbid changes in the valves and the adjacent structures are insidious and gradual. The disease which initiates these is not independent of rheumatism, but is often unaccompanied by pronounced rheumatic phenomena. The endocarditis which results in mitral insufficiency is more violent and more obviously associated with ordinary acute rheumatism; that which induces stenosis is more protracted and symptomless, giving rise to a gradual welding of the curtains and a slow formation of fibrous tissue which, under the even pressure of the blood within the auricle and the ventricle, tends to the production of a smooth septum. This septum becomes gradually thicker, for it has to bear the chief strain of the auricular pressure—not the ventricle, as in the case of mitral insufficiency.

When the acute signs of rheumatic endocarditis have passed away, or when, in the absence of any obviously acute manifestation, the obstructive lesion has been gradually induced, compensation enduring for protracted periods may ensue. Such compensation is a simpler matter than in the case of mitral insufficiency, for an increase of power in the muscle of the right ventricle and of the left auricle only is necessary to maintain it; enhanced force and increased capacity of the left ventricle not being also required as in the structural lesion inducing mitral regurgitation. The left ventricle may deviate but little from the normal, and a strong right ventricle, aided by a hypertrophied, or at least not enfeebled, auricle, will urge a sufficiency of blood through the narrow orifice.

The *symptoms* of failure of compensation differ in many points from those in cases of mitral insufficiency. In the latter the signs are more uniform—the dyspnœa of effort, or the paroxysmal dyspnœa progressively increasing in intensity, the gradual oncome of dropsy, and other signs which have already been considered are evidenced; in mitral stenosis, on the other hand, the symptoms are more erratic, the accidents of the disease predominate, and it is these rather than the gradual heart failure that have in the greatest degree to be reckoned with.

One of the earliest symptoms to attract attention in cases of mitral stenosis is epistaxis; Duroziez has noted this, and I confirm his observations. Probably we are not told of this symptom in many of our cases in hospital because it is considered trivial. In some, though in a less proportion than I should have imagined, there have been complaints that the patient is soon "out of breath." Precordial pain and distress are noted, however, in a considerable number of patients, and in some of these palpitation. My notes show these symptoms in 21 out of 54 cases. Hæmoptysis was recorded in nine of these cases; it occurred in the course of the lung affections in many more. The most frequent of all the induced morbid states is that evidenced by dyspnœa,

cough, and other symptoms referred to the lungs. In some c
is a general bronchitis; but in the great majority there are
localised pneumonia, in the course of which the sputa are f
blood-stained. The bronchitis can be referred to the gener
engorgement of the lungs, but the localised consolidations are p
morbid anatomy as well as by clinical evidence, to be due to i
of branches of the pulmonary artery. These occur with th
degree of frequency in mitral stenosis; and in at least half t
have observed they have been manifested at some time of the li
The hæmoptysis and the lung signs often suggest the prob
pulmonary tuberculosis; but in the vast majority of cases this is
I have mentioned, however, the fact of its occurrence in
minority; so that investigation should be made for tubercle bac
sputum, and the other related signs should be duly weighe
symptoms which occur in the course of mitral stenosis, incre
dangers of the disease and adding new difficulties to its treat
those due to embolic infarctions of the systemic arteries. The
considered in the next group of cases. In only a few cases
clinically observed in the spleen, though morbid anatomy te
this is a very frequent site of embolism. Probably the symp
occasioned pass in many cases unnoticed and unknown. It is
when an intra-cranial artery is thus blocked—then the dang
condition is proclaimed. It is to be remembered that these em
whether in the pulmonary or in the systemic circulation—v
occur in mitral stenosis from detachment of the vegetation
endocarditis, but most frequently from plugs passively formed
chambers of the heart. Frequently, therefore, they are
manifestations of disease, and not symptoms developed during
or subacute illness. They occur both in the cases which are
associated with rheumatism, and those which present no suc
relation. Of course they tend further to disturb compensation,
many cases there is recovery for long periods.

Generally speaking, in the cases of mitral stenosis œdema is
so marked a symptom as in the cases of mitral insufficiency.
and slight œdema occurs in many of them, but general dropsy r
the final stages, when the right chambers of the heart have beco
and the tricuspid valve incompetent; and many patients die b
stage is reached. Sir W. Broadbent notes that great enlargem
liver with true pulsation of this organ is more frequently found
sequence of mitral stenosis than of other valvular affections; an
uncommon to find fluid in the peritoneal cavity before œdema
and legs. The œdema will disappear with rest in bed while ascit
for a time; whereas cardiac dropsy in mitral and tricuspid in
begins, as a rule, in the connective tissue of the most depen
(6). My own observations confirm these conclusions.

In the rheumatic group of cases the influence of sex in the
to the obstructive mitral lesion is well marked and difficult to ex

264 cases of all forms of mitral stenosis collected by Sir Dyce Duckworth, 177 were female and 86 male. In Hayden's cases the proportion of females to males was two to one. In Broadbent's list of 53 cases examined post-mortem, 38 were females and only 15 males. Sir Dyce Duckworth concluded that in 70 per cent of the cases of mitral stenosis tabulated by him there was a certain or strong presumption of rheumatic antecedents; and he considered this estimate of the relation to rheumatism to be rather under than over the mark. In regard to my own cases, in 17 autopsies of children manifesting mitral stenosis in conjunction with pericarditis or endocarditis, which I judged to be of the rheumatic form, 10 were female. Of 35 children under 12 years clinically observed, 22 were female; of 31 adults with mitral stenosis in distinct association with rheumatism observed by myself, 18 were female. It would appear, therefore, that the preponderance of cases in the female sex in my own experience is not so great as in that of other observers. It must be remembered that I have taken those only in which I considered the rheumatic association to be strongly accentuated: the groups of cases not decidedly associated with rheumatism will be considered hereafter.

Prognosis.—I have found the average age at death of 61 patients with mitral stenosis to be 32·7. The late Dr. Hayden's cases—42 in number—gave an average age of 37·8. Sir W. Broadbent states that the average age at death, deduced from 53 cases abstracted from the post-mortem records at St. Mary's Hospital, was 33 for males and 37 and 38 for females; and he adds: "Mitral stenosis stands next to aortic regurgitation among valvular affections in the order of gravity." I have records of 17 cases fatal before the age of 12 years, the average being 9½ years; 10 of these at the age of 10. The association with rheumatism is shown by the fact that, in addition to the valve-lesion, in 14 of these either pericarditis or recent endocarditis of rheumatic characters were found in necropsy. The rheumatic associations of the majority of cases of mitral stenosis constitute a very great, if not the chief element of danger. The other causes of fatality will be pointed out in the consideration of the other groups. It must be accepted as a general proposition that the subjects of mitral stenosis (discovered at an early age) rarely survive the age of 40; the disease, therefore, when dating from childhood and adolescence, and in such cases having its origin, as I believe, in a rheumatic affection, is of grave significance.

Treatment.—The recognition of the rheumatic association of mitral stenosis is of much importance in treatment. In childhood and adolescence a slight febrile attack in the subject of mitral stenosis, or in one who presents signs of the advent of the lesion, should be held as a probable indication of a subacute rheumatism; and treatment by complete rest with the administration of salicin or the salicylates should be enjoined. If cough and difficulties of breathing are also present, symptoms of bronchitis or pneumonia, the systematic administration of ammonia in addition is valuable. The frequency of infarction of branches of the pulmonary artery in cases of mitral stenosis has already been pointed

out. The late Sir Benjamin Richardson advocated the frequent
tration of liquor ammoniæ, well diluted, as a means not only of
fluidity of the blood, but also of dissolving a coagulum already
There may be differences of opinion whether such solution
possible; but there are many reasons in favour of the treatment.
increasing the alkalinity of the blood-plasma, ammonia is a
stimulant of the nervous mechanism of the heart and of the res
centre; and, by increasing the bronchial secretion and rendering
fluid, it acts very favourably as an expectorant. I have wit
case in which death seemed impending from plugging of the pu
artery when the frequent administration of ammonia seemed t
means of saving the patient's life; and I have observed many
which there were signs of partial embolism when recoveries w
satisfactory. The best mode of administration in young subjec
liquor ammoniæ fortior in doses of one to five minims, wit
extract of liquorice well diluted with water; the dose being re
according to the urgency of the case—every half-hour, every
every two hours until signs of improvement appear. It may
continued every four hours for several days. Whether there be b
from venous congestion or local consolidations of the lungs from
the ammonia treatment is valuable. It may be well to issue a
against the use of digitalis during any febrile manifestation in the
I have found it worse than useless. The hæmoptysis which m
should not be treated by styptics or opium. As a general r
better that any hæmorrhage which breaks out in the course
stenosis should not be checked by drug treatment. A like
treatment to that just mentioned may be put in force in cases
precordial pain or distress is manifested in the subjects o
stenosis. It is to be remembered that pericarditis arises not infr
in this connection, when the special treatment for this disease
carried out. The occurrence of pericarditis or of lung complic
any kind may rapidly break the compensation in mitral steno
inadequacy of the right heart, with dropsy and other signs
failure, may occur. In such case the treatment should be as
described under mitral insufficiency. The symptoms, howe
frequently recovered from, and compensation is restored.

Whilst there are any indications of acute changes—of end
of pericarditis, of rheumatism, or of any symptoms attended by
—perfect rest in bed should be enjoined. It is otherwise, how
convalescence, when it is to be presumed that sclerosing chang
valve structures are going on. Then systematic exercises, gra
tentative at first, should be recommended, for they fulfil in
indications; they not only aid the venous circulation, but by ex
the thorax they tend to aspirate the heart, increase the outfl
auricle to ventricle, and perhaps prevent the imminent dange
progressively increasing contraction of the auriculo-ventricular
It may be urged that a danger of such exercises may be a detac

a vegetation left by the rheumatic endocarditis; this is possible, but it is proved that the greater danger is the passive formation of thrombi within the heart in consequence of retarded circulation within it. The patient should be cautioned against violent movements, but there can be no doubt, I think, of the value of systematic exercises. These have been considered in the treatment of mitral insufficiency. During convalescence from any acute febrile manifestation in the subject of mitral stenosis, the first method employed should be gentle massage and movements of the legs, the patient being quiet in bed; next in order the arms and thorax. Later, expansion movements of the thorax, made by the patient himself cautiously and deliberately, should be practised, with judicious intervals of rest. Concurrently, or, as I prefer, just subsequently to these movements, there should be spongings, first with warm and afterwards with cool water, followed by dry towel friction. Later the systematic muscular exercises, as prescribed by Ling and Schott, may be used. An excellent summary of methods of muscular movement is given by Dr. Lauder Brunton (7).

Although moderate exercise in the fresh air in the subject of fairly compensated mitral stenosis is salutary, sudden overstrains are dangerous. In some cases breathlessness does not come as a warning, and patients persist in overtaxing their strength. The subject of mitral stenosis should be protected from chills by suitable apparel, and no clothing is better than pure woollen. A light woollen night-dress is also to be recommended. Heavy overcoats and sealskins, which weigh down the shoulders and thus prevent good expansion of the thorax, are, in my opinion, to be deprecated.

The late Sir Andrew Clark, in a clinical lecture which was published after his death, gave some valuable hygienic rules for a patient with mitral stenosis. In the daily dietary fluids should be restricted, for after their absorption they distend the vascular system, and increase the bulk without increasing the nutritive value of the blood within the vessels. The ingestion of much liquid enfeebles the heart and increases the labour of the right ventricle and left auricle in the transmission of blood through the narrowed aperture into the left ventricle. The patient should have three good meals a day as dry as he can make them; over-eating and indigestible foods must be strictly guarded against. I consider it a good plan to advise that the two meals of the day of which meats form a portion should be taken without alcohol, and with a little pure water or toast water only; and subsequently to each of these a wineglassful of milk with two teaspoonfuls of good old brandy or whisky may be allowed. In some patients there is a slight appearance of jaundice, the liver is embarrassed; there is often constipation. There may be basic congestion of the lungs. Sir Andrew Clark said, "To relieve the lungs give something to relieve the bowels." Sulphate of soda and phosphate of soda, equal parts in powder, may be administered in doses of two or three teaspoonfuls dissolved in water in the morning, or a teaspoonful of sulphate of magnesia may be taken in hot water. Such aperients relieve the portal system, and so the right earliest changes. At any rate the only cause concerning which we have precise evidence is rheumatism.

The most characteristic among the severe lesions of the nervous system is right hemiplegia. In one of my cases, a girl of 10, the first detected sign was sudden paralysis of the right arm and leg; the child recovered completely from the paralysis, but died seven months afterwards after having manifested much precordial distress. Mitral stenosis was demonstrated at the autopsy, and there was universal adhesion of the pericardium. In another patient, a woman aged 22, who had never manifested any symptom of rheumatism, and who had no hereditary tendency thereto, sudden right hemiplegia occurred with aphasia. There were pronounced physical signs of mitral stenosis without regurgitation. The patient made a perfect recovery from the paralysis of motion, but complete aphasia persisted (28). In another of my cases, also a woman, left hemiplegia occurred; after full recovery from this lesion right hemiplegia came on suddenly; from this latter attack the recovery was but partial. In Duroziez's 43 cases of "pure" mitral stenosis in females, 11 manifested right hemiplegia with aphasia, and 4 hemiplegia without aphasia; there were no such cases in the male sex.

Another nervous disorder which may suddenly arise in subjects of the affection is hemichorea. In 38 cases of mitral stenosis I found 4 of hemichorea. Duroziez records a case of a woman, aged 24, with mitral stenosis declared by right hemichorea in which the convulsive movements of the limbs ceased, but chorea of articulation remained, so that the beginning only of each word was uttered. One of my patients, a boy aged 3½, was suddenly seized with epilepsy, the unconsciousness lasting twenty minutes. Nine months afterwards chorea became manifested; recovery took place, but after a second period of nine months another attack of chorea occurred; there were well-marked physical signs of mitral stenosis. In a boy, aged 5, who manifested presystolic murmur and thrill, a fit had occurred eighteen months previously attended with unconsciousness so profound that the child was thought to be dead; nine months afterwards chorea appeared. In another case, a girl aged 5, epilepsy occurred, and the attacks were repeated and severe. In a lad, aged 18, in whom I had the opportunity of watching the physical signs of the gradual establishment of mitral stenosis, from the manifestation of a soft apical systolic murmur to that of complete and characteristic presystolic murmur, thrill, and doubled second sound, there occurred during his exercise in the garden a sudden unconsciousness, which was complete for a minute or two, but was not attended by muscular spasm.

It is, I think, reasonable to conclude that these sudden perturbations of the nervous system are caused by infarctions of branches of the intracranial arteries; in some instances this was positively proved by necropsies. It is clear that the consequences of such embolism may in some cases pass away completely; in others the plugging of the vessel is followed by necrosis of the nervous structures thus supplied.

consolidations may be found in the lungs—the group of symptoms closely resembling those of pulmonary tuberculosis. In the great majority the diagnosis of pulmonary consumption is not justified; the symptoms are the accidents of the mitral disease itself. I have given my reasons for dissenting from the view that mitral stenosis can be considered as standing, even remotely, in any causal relation to tuberculosis; but I think it probable that the deficient arterial supply which is a consequence of the disease disposes to the occurrences of tubercular changes in the lungs in a small minority of cases, and the remote probability of this should be present in the mind of the observer. The presence or absence of tubercle bacilli in the sputa will settle the question.

In the treatment of this group of cases physical training should hold a first place. It is evidently of the highest importance that the blood-flow from the right to the left ventricle should by judicious means be increased. It is possible that if this be accomplished by systematic muscular movements and careful hygiene at an early period of the manifestation of the morbid condition, the insidious contraction of the orifice may be averted. The means to this end are frictions, massage, carefully planned muscular movements, baths and bathing, the selection of suitable climates, and the regulation of diet. Medicinally iron, arsenic, small doses of liq. strychninæ, and cod-liver oil are the chief agents to be employed. The treatment of complications and of failure of compensation will be as in other groups of the disease.

Group IV. Cases associated with chronic renal disease and arterio-sclerosis.—As I have already stated, the association between mitral stenosis and chronic renal disease was first pointed out by Goodhart (11), and confirmed by Pitt in 1887. The observations were made chiefly from the standpoint of morbid anatomy, though Pitt contributed some clinical data. It was made clear that the cases demonstrating the coexistence of the two morbid states are by no means infrequent. Nevertheless Gérard and others hold that mitral stenosis, having its origin in arterio-sclerosis, is rare. I cannot doubt that the explanation of this apparent conflict is to be found in the fact that the cases demonstrating the conjunction of the diseases are most frequently found after death; they come under clinical observation with comparative rarity. The two morbid affections progress insidiously, and either the patient is suddenly stricken down with apoplexy, or some sudden complication which precludes any physical examination, or, if such examination has been possible, the physical signs were supposed to indicate some form of disease other than mitral stenosis.

I have notes of six cases observed by myself, in which, without any evidence of rheumatism or other predisposing malady, there have been signs which should bring them into the group under consideration. In three other cases aortic valvular disease was conjoined with the mitral. Several others could be regarded as mixed cases, these having rheumatic antecedents; but the subsequent evolution was after the manner of arterio-sclerosis. In my cases the most advanced age was 70, the only

male; the youngest was 35. The cases recorded by Blind (
Gérard (five) which should come into this group are nine in nu
men and four women, the oldest patient 67, the youngest 32
cases taken as examples there is no rheumatic antecedent; b
majority the usual signs of chronic Bright's disease are pres
radial and other arteries are firm and incompressible; the us
trophy of the left ventricle of the heart, however, is not manif
some of the cases, in addition to the signs of thickened arteries,
obvious evidences of gout with deposits of urates in the joints
where. In some there are well-marked signs of arterial atheron
may be emphysema of the lungs or pulmonary fibrosis. Fibro
may occur about the viscera, the periviscеritis of Huchard. T
of the disease is not to be traced, the progress is slow and imp
The physical signs of mitral stenosis in many of the cases do
from those ordinarily observed—the presystolic murmur, the entir
or the early or mid-diastolic murmur, the sudden, loud first sour
double shock sound in diastole. In some cases there is no
murmur, but a systolic. This may be heard at the apex and
thus answering to the criteria of mitral regurgitation; in sucl
probable that the auricle has become dilated and weak. The
of stenosis can only then be made from the evidence of a he
enlarged right ventricle, contrasting with the absence of signs
ment of the left ventricle, perhaps also from the absence of a
sound at the apex (Broadbent). Exceptionally there is no lo
short, sudden first sound, but a dull sound as in the case of
trophied left ventricle. In the cases manifested between the ages
40, there have been the evidences of the gradual oncome of ch
disease with thickened arteries or undoubted gout with deposits
There is no evidence of any pre-existing disease of the val
rheumatic or other causes; but there must be a remaining doul
any change in the valve preceded the fibrous proliferations intri
Bright's disease. It is, I think, improbable, seeing that the grea
of cases due to rheumatism are fatal before the age of 40, th
Bright's disease is a superadded factor, for if so the scene woul
speedily closed, and death would ensue. It is at first sight m
that the changes are independent of rheumatism and due to a
of sclerosis.

In one case, that of a lady aged 52, I had opportunity of
the gradual involution of the disease as evidenced by the phy
and confirmed by post-mortem examination. There was at fir
whatever of cardiac disease, but gradually all the usual signs
stenosis were manifested. The urine showed normal characters
the whole period of observation, and the case was observ
thirteen years. The symptoms were those of dyspepsia, wit
implication of the nervous system, first evidenced by an epile
and afterwards by dementia. The necropsy showed funnel trans
of the mitral valve, with much fibrous thickening of the su

structures. There were chronic fibroid thickenings in the pleuræ, the left lung, the spleen, the liver, the capsules of the kidneys, and the membranes of the brain. The granular changes in the kidneys were but slightly pronounced, and no doubt comparatively recent. The chief morbid change was the widely-spread fibrosis, the progress of which had been very gradual; and it seemed legitimate to infer that the stenosis of the mitral orifice and the fibrous transformation of the surrounding structures were due to a similar morbid process. This case is no doubt exceptional in that the fibroid changes in so many situations long preceded any signs of interstitial nephritis. In the majority of cases the evidence of chronic renal disease is well marked when the case comes under observation. The age 50 to 70 renders it improbable that obstruction of the mitral orifice from any cause had preceded the gradual evolution of the chronic renal disease with its attendant arterio-sclerosis. In all cases the progress of the disease must have been very gradual and insidious. In many there have been signs of cerebral disease; indeed, it is for symptoms indicating such disease that the cases usually come under notice. Epileptiform seizures, apoplexy, dementia, or uræmia, are the chief forms. In several instances the signs of albuminuria retinitis have been recorded. That the morbid changes have been slow and gradual is shown also by the post-mortem evidence. In the case of a woman of 65 there has been found a funnel transformation of the mitral curtains; just as observed in the cases in earlier life; but in the majority the button-hole form of mitral stenosis is manifested with great thickening and firm fibrous transformation of the papillary muscles.

The *treatment* in this group of cases is subordinate to that of the chronic renal disease and the attendant thickenings of the arteries. It is important to realise that the prognosis is very grave. When a patient manifesting the signs of mitral stenosis at whatsoever age presents signs of firm and thick arteries, and the urine is found to be continually of low specific gravity and occasionally albuminous, it is well that for a few weeks an entire milk dietary be enjoined. It may be a little difficult to convince a patient past middle age, whose stomach has been the receptacle of foods of many and various kinds far more than adequate to the needs of his organism, whose nerves of taste have been frequently and abnormally stimulated, and whose absorption of nutritive material and excretion of effete products have been after the manner of periodic and irregular thunder-showers which have deluged the land and blocked the drains, that he must return to the sweet simplicity of the earliest months of his life. Yet it is best so. It is the absence of the irritation to the arterioles caused by the complex albuminoids which turns the balance towards amendment. It may be necessary, however, to make some concessions. In the early morning, or on waking, the patient may take half a pint of milk with half an ounce of rum, or of cognac and an ounce of lime water. In some cases one to two ounces of fluid magnesia may be substituted with advantage for the aqua calcis. Three or four hours afterwards a second half-pint of milk may be taken flavoured with a little hot coffee; the third

half-pint, after a like interval, may be taken as a blancmange
isinglass or gelatine. At similar intervals, during the remain
waking hours, the changes may be rung with the various fl
but no solids should be permitted other than light biscuits.

The total amount of milk taken in the twenty-four hours
three to six pints. The total quantity of cognac or spirits o
should be limited to two ounces. To break the monotony of
milk diet, it is a good plan to allow occasionally a firm
flavoured with madeira, rum, kirschwasser, or chartreuse. O
tablespoonfuls of isinglass are to be melted in very hot wate
milk added thereto; the small quantity of gelatin thus mingle
milk is sufficient to prevent any firm curdling of the casein in t
the coagulum being rendered much softer and its digestion facili

In regard to medicinal treatment, the rule of Dr. G. W. Bal
be followed, that no cardiac tonic should be administered
simultaneous unlocking of the arterioles. The therapeuti
should follow the lines already described in the consideration
II. of cases of "Mitral Regurgitation." As Sir Wm. Broadbe
"Nitro-glycerine and other vaso-dilators may sometimes be
good effect for many weeks or even months in conjunction w
tonics, such as iron, quinine and nux vomica" (5).

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